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CORN STATES

in Production Methods

Unloading virus pigs at the "Corn States" plant.

Virus pigs used in "Corn States" production are procured by direct shipment from areas where raising for the fat hog market is exceptionally rare. Exposure in centers of livestock handling is not allowed in transit. Where shipment is by railroad, a company owned switch permits unloading without passage through public stockyards.

FURTHERMORE,

All "Corn States" simultaneous virus is selected following an examination of cultures of individual bleedings of cholera-sick pigs. Serial lots of virus are examined bacteriologically and subjected to the pigeon test for Erysipelothrix rhusiopathiae. Also, each serial is required to satisfactorily pass an adequate virulency test for hog cholera.

All of these production safeguards are designed to implement the following statement:—

The use of anti-hog-cholera serum and virus should be restricted to qualified professional hands. — *M. Dorset.*

THE

CORN STATES SERUM COMPANY

OMAHA

Journal
of the
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Vol. XCV SEPTEMBER, 1939 No. 750

Incoördination of Equidae: Wobblers*

By W. W. DIMOCK, D.V.M., and B. J. ERRINGTON, D.V.M., Ph.D.

Lexington, Ky.

DURING the past year, in this district, there has been an apparent increase in the occurrence of an equine disease characterized by a bilateral incoördination of the hind limbs. Animals thus affected are called "wobblers." The disease has existed for many years and has not been confined to this state. In some localities an apparently similar disease has been descriptively termed "jinxed back" and "weak loin." Perhaps because of a general belief that it was the result of an injury and also because of its sporadic appearance, little has been reported concerning this disease.

For several years, on some farms, the disease has existed with low incidence, but in many cases the incidence on a farm in one year seems to have little significance as to the number of cases that may occur the following year. The highest morbidity on one farm in this district occurred in a stud of 53 mares, among which there were two cases in 1936, two in 1937 and seven in 1938. The following report covers the period from May 15, 1937, to July 1, 1939. During that time we observed 47 cases and autopsied 30 cases of wobblers.

AGE INCIDENCE

The disease appears in sucklings, weanlings, yearlings, and occasionally in 2-year-old animals. It rarely, if ever, occurs in older horses. Six of our cases occurred in sucklings, ten in weanlings, 26 in yearlings and five in 2-year-olds. The youngest animal was 3 months old when the symptoms first appeared. It is very common for the symptoms to appear near weaning time, which in Thoroughbreds is usually during September and October. Nine of our cases in sucklings and weanlings occurred during these months. On one farm the cases, over a period of years, have always appeared at weaning time. Two of the four 2-year-old animals had been suspected of being abnormal because of their unusual gaits as yearlings. Only three of the 47 cases appeared during the months of December, January, February, and March, and at least one of these had previously shown an abnormal gait. The highest monthly inci-

*The investigation reported herein is affiliated with a project of the Kentucky Agricultural Experiment Station and is published with the approval of the director.

From the department of animal pathology, Kentucky Agricultural Experiment Station.

dence was nine cases, which occurred in July, 1937.

INCIDENCE AS TO BREED

Of the 47 cases, 38 were colts and nine were fillies. The morbidity among Thoroughbred and Saddle-bred animals is proportionally higher than among other breeds of horses in this district. Although there are many Standard-bred horses born and raised here, it is questionable whether or not the disease has ever occurred among them. One of the cases observed was a Percheron filly and two were mules. The condition has also been observed in farm-bred work colts.

SYMPTOMS

The onset of the symptoms is often sudden. They are always bilateral and quite typical, although this may not always be true when the first symptoms are seen. Bilateral symptoms sometimes may be so slight that their detection is difficult. In the case of several yearlings the first symptom was a weakness shown only under saddle. The animals seemed to stagger under the extra weight. Within a week or ten days each of these showed typical incoördination without the extra weight. Another yearling, which showed lameness in one hind leg and in the opposite leg the following day, did not develop typical incoördination until a week later. Frequently, an animal appearing normal in the morning when turned out in pasture is found at the end of the day with typical symptoms, which may even be extreme.

The symptoms vary from a slight incoördination in the hind limbs, which may be evident only under certain conditions, to an ataxia of all four limbs and restricted movement of the neck. The attitude of the animal when standing is normal except in extreme cases, although sway back tends to develop in the course of months. An animal with slight involvement may walk steadily but overreach with each step. There is no tendency for the animal to shorten its stride. Some show no unsoundness when walking except in turning, when a slight incoördination in the hind parts or knuckling over of the feltlock is evident.

An animal with comparatively mild symptoms is able to trot and gallop surprisingly well, although, when galloping, the hind limbs seem to be carried with lessened tension and appear to swing forward from momentum rather than from muscular effort. It may be little hampered in these faster gaits until it stiffens the legs in an attempt to stop, or is forced to turn abruptly. It then invariably shows knuckling over in a hind fetlock, a hitching, or a dropping of the hips as from a sudden relaxation of all the extensor muscles of the limb and, consequently, is unable to stop abruptly or turn sure-footedly. A mild case may be diagnosed only by observing these symptoms in stopping or turning.

Although the salient symptom is the incoördination in the hind parts, the fore limbs are involved in a majority of cases. The peculiar wobbling in the hind parts causes difficulty in determining if and when the fore limbs are actually involved. There is a tendency for the fore limbs to be carried wide when moving, as if to aid in balancing the body. Mild or extreme symptoms involving both the fore and hind limbs may be present at the onset. However, more frequently the symptoms are shown in the hind limbs for a period varying from days to weeks previous to the involvement of the fore limbs. The ataxia, which may be slight when only the hind limbs are affected, becomes more extreme when the fore limbs are definitely involved.

The incoördination may be of such degree that, when moving, the animal wobbles unsteadily in all directions, as might be expected from acute alcoholism. It did not seem safe to approach one of our most extreme cases, a 2-year-old filly, because of her unsteadiness whenever she moved, although she was always able to stay on her feet. The ability to get up and down is retained until the ataxia is very advanced or the animal has worn itself out from struggling while down without making a concerted effort to get up. Development of symptoms to this advanced degree is the exception rather than the rule.

Clinical symptoms are often seen in the cervical region. Flexing the neck to one side or the other causes a manifestation of pain in many cases and, when the animal moves, there is a tendency for the head and neck to be carried extended. Protuberances, apparently caused by the transverse processes of the cervical vertebrae being out of alignment, are sometimes evident. This was most prominent in the youngest affected animal, which was autopsied six months after the onset of symptoms (fig. 1, B). This was the only animal in which wasting of the neck muscles was evident. Slight pressure over the tuber coxae or lumbar vertebrae in advanced cases frequently causes more than a normal flinching that, from day to day, may shift from one location to the other or be entirely absent.

Other sensory disturbances and fecal and urinary incontinence have not been observed. In severe, acute cases there may be a slight rise in temperature which is probably due to pain. Only one animal, a weanling, which was treated for intestinal toxemia a week before developing wobbler symptoms, had shown any previous general symptoms. All of the cases were animals in good condition. Often they were the animals that were doing the best in their group. After the onset of symptoms their development may be restricted or not, depending upon the extent of the symptoms.

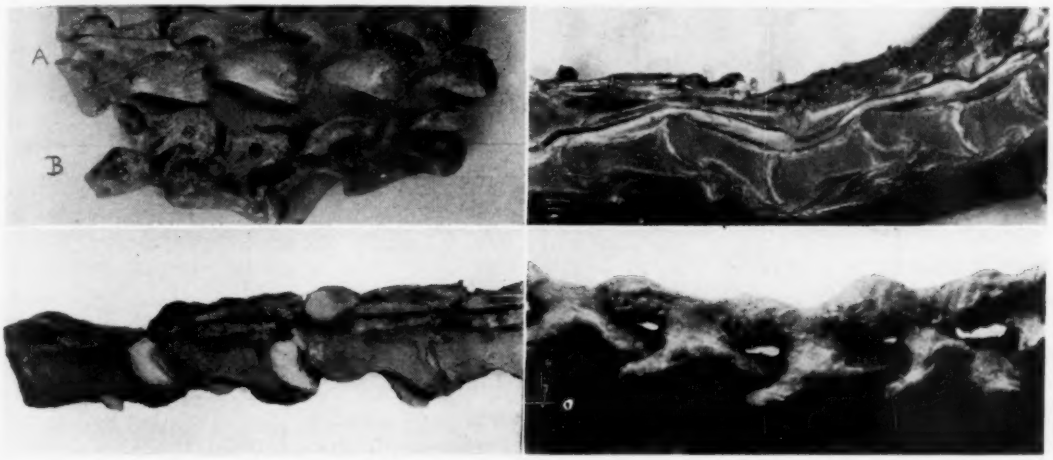

Fig. 1 (upper left). A: Third, fourth, fifth and sixth cervical vertebrae of a yearling colt, taken from the left side. Anterior articular process of fourth cervical vertebra in contact with body of the third cervical vertebra. The constricted foramen and the cause of restricted movement of this articulation are evident. Figure 1A and figure 3 represent the same animal. B: Third, fourth, fifth and sixth cervical vertebrae of a weanling colt, taken from the right side. Anterior articular processes of the fourth and sixth in contact with the bodies of the third and fifth, respectively, causing constrictions of the spinal foramen. Figure 2 represents the same animal.

Fig. 2 (upper right). Curvature and constriction of the cervical spinal canal. The pressure on the spinal cord is evident. Irregularities which were present in the anterior epiphyseal line of the vertebral bodies, apparently due to a change in the normal pressure from adjoining vertebrae, are not clearly shown.

Fig. 3 (lower left). Articular surface anterior process of fourth and fifth cervical vertebrae exposed. Note comparative shape and position of the processes. The articular cartilage of the fourth cervical vertebra is very thin, dark, and erosion extends into the subchondral bone.

Fig. 4 (lower right). Curvatures involving the spinal canal and constriction of spinal foramen are shown. Both specimens were obtained from yearling colts.

PROGNOSIS

Though the prognosis for a complete recovery is always unfavorable, the extent to which the symptoms will develop can not be predicted in the early stages. Any change in degree of incoördination, either an increase or decrease, is apt to occur within the first few weeks after the onset. Any change after that is usually very gradual. In many cases the first symptoms of slight incoördination in the hind limbs may not increase, even though the animal

be kept for years. One 11-year-old Thoroughbred, in which the symptoms have not changed to any extent from their first appearance, has been a satisfactory brood mare since being retired at that time. Another stallion with a similar history has been successful in service for several years. A few animals show improvement in time. In other cases the fore limbs become involved; the incoördination progresses in spite of all treatment until the possibility of recovery is given up by all concerned. Insurance companies in this area do not hesitate in paying the insurance on an animal diagnosed as a wobbler.

THEORIES OF THE DISEASE

Many of the affected animals have histories of injuries in pasture, such as running into a fence or falling into a watering trough, after which they immediately showed symptoms typical of wobblers. The frequency of such happenings has supported the belief that injury is the common cause, whether or not there is evidence or history of any traumatism. However, the number of animals affected and the fact that few, if any, ever recover after showing symptoms, suggest that injury is probably not the causative factor of wobblers. Neither fractures nor dislocations of the spinal column have been found, although it is conceivable that such injuries may cause symptoms similar to those of wobblers. The conclusion that wobblers would not have been involved in these accidents if they had previously been normal is supported by such histories as inability to carry weight, stiff neck, or slight abnormalities in the gait being observed previous to the development of typical incoördination.

The appearance on the same farm of several cases in one group, and none in separate groups under similar conditions, and the fact that some large farms have never had a case, have caused some observers to suspect the disease to be infectious. Some have suspected nutrition for similar reasons. Wobblers out of the same mare in succeeding years have been reported as occasionally occurring. This happened in one instance among our cases. We have observed no other close relationship among these cases.

INOCULATION EXPERIMENTS NEGATIVE

We have been unable to reproduce the disease by injections of cerebro-spinal fluid, emulsion of spinal cord, emulsion of the brain, or a mixture of these from affected cases into the subarachnoid space, the spinal cord or cerebrum of horses of susceptible age. Two of the cases from which two weanlings and two yearlings were injected had shown symptoms for less than one week at the time of autopsy. Five other injections were made into yearlings, with material from cases that had shown symptoms for eight days to five weeks. Direct and indirect blood transfusions from seven cases of wobblers of less than one-week duration produced no symptoms in one weanling and six yearlings.

SPINAL AND CERVICAL FLUIDS STERILE

No bacteria were isolated from the spinal fluid or from the synovial fluid of the cervical and limb articulations. On those culture tubes inoculated from the bone beneath the articular cartilage of the cervical vertebrae, where growth occurred, it was due to *Bacillus proteus* or a spore-forming organism. The blood-serum calcium and inorganic phosphorus were within the normal range in acute and chronic cases.

RELATION OF LESION AND SYMPTOM

The symptoms shown by "wobblers" indicate lesions interfering with the functioning of the spinal cord. Degenerative changes were found in the cervical and lumbar regions of the spinal cord. Microscopic lesions were present in the spinal cord of cases in which no gross lesions were observed. Although a study of the sections taken from the spinal cord of these cases has not been completed, it seems probable that degenerative changes will be found in a significant number of them.

The carriage of the head and neck and the symptoms shown on clinical examination indicate lesions in the cervical region

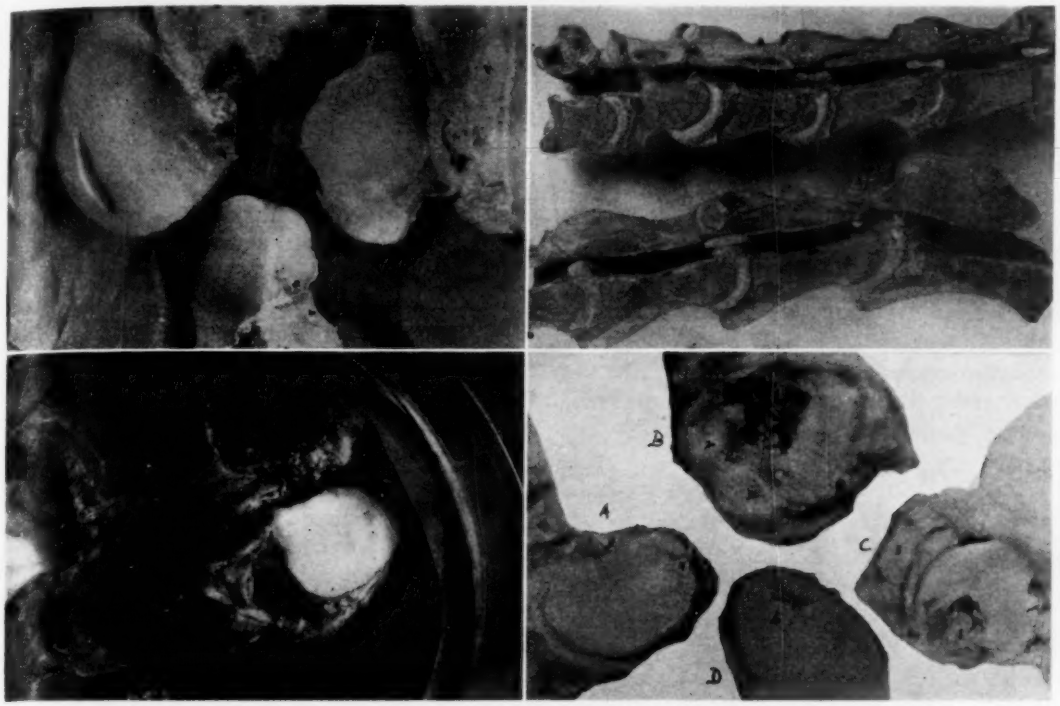

Fig. 5 (upper left). Hypertrophy of bone and cartilage involving spinal foramen. 1) Third cervical nerve; 2) spinal canal; 3) spinal foramen; 4) spicules of bone; 5) irregular formation of bone and cartilage.

Fig. 6 (upper right). Hypertrophy involving articular processes between fifth and sixth, and sixth and seventh cervical vertebrae of a weanling colt. Note comparative size of spinal foramina.

Fig. 7 (lower left). Anterior articular process from a weanling colt. Between the margin of cartilage and attachment of the capsule is an area of irregular, rough bone covered by a thin membrane, in which is a spicule of bone (1).

Fig. 8 (lower right). A, 4: Pedunculated mass of cartilage connected to joint capsule by a strand of fibrous tissue. A, 5: Strand of fibrous tissue from capsule to irregular mass of cartilage. (The figures are on a wedge-shaped piece of paper placed beneath the strands of fibrous tissue.) B, 1: Dense connective tissue from subchondral bone. B, 2: Shelf of hypertrophied bone covered with cartilaginous tissue. B and C are opposing articulations and were ankylosed due to the fibrous tissue (1). C, 1: Same as B. C, 3: Mass of cartilage in the joint capsule. A, B and C were obtained from a yearling that had shown symptoms as a weanling. D, 6: Shelf of cartilage, raised from subchondral bone, open on one side. There were four free masses of cartilage in this joint. This is a specimen from a weanling that was affected as a suckling.

in many cases, especially those in which the incoördination is more than slight in degree. Constrictions of the cervical spinal canal, which were present in eleven cases autopsied, varied from a slight upward curvature of the floor of the canal at one or more of the intercentral articulations (fig. 6) to a constriction measuring one-third of the diameter of the canal at adjacent articulations (fig. 2).

The cervical spinal canal was constricted laterally in some cases from encroachment of the anterior articular process, which curved downward to contact or form a false joint with the body of the preceding vertebra (figs. 3 and 6). This contact between the articular process of one vertebra and the body of an adjacent one would be expected to cause the restricted movement of the neck which these cases show. Malformation of the articular processes caused constriction of related spinal foramen (figs. 1, 3 and 6). Constriction of the foramen was due in other cases to hypertrophy of bone or bone and cartilage involving the articular process (fig. 5). Degeneration of cervical spinal nerves was found. In advanced cases the nerve bundles consisted largely of tissue, often arranged in con-

centric rings, with few normal nerve fibers remaining (fig. 9).

Arthritis involving the interneural joints and osteoarthritis of the cervical vertebrae were commonly present. Mild lesions consisted of occasional shallow clefts, areas of roughness and small areas of tufts of cartilage projected from the surface, all of which were sometimes found in otherwise normal-appearing cartilage. More extensive lesions were present in the margin of the articular cartilage. These consisted of separations between the margin of the cartilage and the attachment of the synovial membrane and peripheral erosion of carti-

Fig. 9. Sixth cervical spinal nerve from a weanling colt which showed the first symptoms at 3 months of age. It was autopsied six months later.

lage. The surface of these denuded areas was irregular and appeared dark red; spicules of bone were frequently present in them (fig. 7). The remaining misshapen articular surface appeared normal in some, while in others it was dull, dry, streaked and more or less rough.

Fibrous tissue from the region of the attachment of the capsule, usually along the anterior margin, extended on to the surface of some articulations with little or no change in the joint other than a dull, streaked cartilage. Dense bands of fibrous tissue, in other cases, passed into deeply eroded areas. In a few cases the degeneration had extended into the subchondral bone on apposing surfaces; dense fibrous tissue connected these surfaces causing ankylosis (fig. 8 [1]).

On the margins of the articulations there was evidence of hypertrophy of bone and cartilage, which in many cases was sufficient to cause contact with the bodies of adjacent vertebrae. The hypertrophy often was evident before opening the joint capsule (fig. 4). In one case of long standing there was a great increase in connective tissue surrounding the joint capsule of a hypertrophied articulation.

Pedunculated masses of cartilage, to which fibrous tissue was sometimes attached, were not infrequently found. Cartilaginous tissue was present in one case, in the fibrous tissue of the capsule (fig. 8 [3]). In another case a shelf of cartilage, unattached to the bone beneath and open at one end, was present. In this joint were four free masses of cartilage of varying size (fig. 8, D). Joint mice were found in other cases.

Less extensive lesions, similar to those in the cervical region, were found in the first few thoracic vertebrae. A different type of lesion, resembling degenerative atrophy and probably not peculiar to wobblers, was present in the tubercle facets of the thoracic vertebrae. It consisted of a smooth depression, which appeared dark red due to the lack or absence of cartilage, extending over as much as one-half of the articular surface and surrounded by normal cartilage. No similar lesions, other than areas of thin articular cartilage, were observed in the joints of the limbs. It was interesting to observe that in three of the cases autopsied, the first rib was missing. There was no facet on the seventh cervical vertebra for articulation with the first rib.

DISCUSSION

While the lesions in the cervical vertebrae probably are not directly responsible for the typical incoördination of wobblers, other clinical symptoms which are frequently present in this region indicate a direct relationship to these lesions. The extent of the osteoarthritis and malformations found on autopsy gives mechanical and pathological cause for the neck being carried extended, pain on lateral flexion, and the bony protuberances.

In some cases the extent and severity of

the lesions did not correspond with the clinical symptoms shown in this region. The fact that the pathological lesions in equine arthritis are not always in accord with clinical symptoms has been frequently recognized. However, in considering the 23 cases autopsied it can be stated that the degree of spondylitis and spondylarthritis of the cervical region, in general, corresponded with the symptoms and the duration of the disease. The lesions in cases of mild arthritis were similar in type to those in severe arthritis.

Degeneration of cervical spinal nerves was found in some acute cases in which there were no related gross lesions, other than a possible acute arthritis. In many cases the constriction of the foramen from the increased connective tissue surrounding the joint capsule, and the osseous malformation and hypertrophy involving the spinal foramen, were indications of a possible cause of the nerve degeneration. It is possible that the spinal nerve degeneration occurs, in some cases at least, as a result of a primary degeneration of the spinal cord. Mitchell,[1] who has studied the osteoarthritic changes in the vertebral column and limbs of aged horses, found an accompanying peripheral nerve degeneration. He has stated the impression that "osteoarthritic lesions arise as a generalized state in which peripheral nerve degeneration is a coincident phenomenon."

In humans the spinal cord may be relatively easily damaged in injuries to the cervical spine, as from momentary nipping, when there is displacement of the cervical vertebrae or momentary stretching over the vertebral bodies.[2] In eleven of the cases autopsied there was curvature with more or less constriction of the cervical spinal canal. Whether the development of incoördination resulted from pressure or stretching of the cord is problematic. Bony protuberances in clinical cases and spinal canal constrictions on autopsy were seen in cases where the symptoms were not of long duration.

CONCLUSIONS

The constriction of the cervical spinal canal and foramen, the spondylarthritis, the degenerative changes in the spinal cord and in the spinal nerves, which have been found in equine wobblers, warrant consideration as significant factors. The correlation between them and their relative importance to the disease is uncertain. Some hope lies in a chemical analysis of the bone, a further histopathological study of the bones, nerves and cord, a correlation of these with the clinical picture, and a more thorough knowledge of the nutritional requirements of Equidae.

References

[1]Mitchell, W. M.: Jour. Comp. Path. & Ther., xlv (1937), p. 283.

[2]Walshe, F. M. R., and Ross, J.: Abst., Arch. Neur. & Psych., xxxviii (1937), p. 1318.

Arthritis Deformans in Colts

From a study of the literature and from his own researches into the racial and family pathology of horses, Schäper concludes that the arthritis deformans of colts, sometimes termed "habitual lateral luxation of the patella," is merely a hereditary disease, the recessive character of which he considers proved. Consequently, the ailment can be combated solely by proper breeding hygiene, that is, the exclusion from breeding of stallions and mares proved to be tainted thus, and the avoidance of all inbreeding, including linebreeding, within such tainted families and lines.

Very exceptionally a colt may partly recover from the arthritis deformans. Such horses may be used for work, but under no circumstances should they be used for breeding. *(Beitrag zur Vererbung der Kniescheibengelenkentzündung bei Fohlen (Hereditary transmission of arthritis deformans in colts), by W. Schäper. Tierärztl. Rundschau, xlv (1939), p. 7.)*

There are 2,205 physicians in the United States who limit their practice to pediatrics. Of these, 1,006 are certified by the American Board of Pediatrics; and 2,166 general practitioners have declared themselves as especially interested in pediatrics.—*Edward Clay Mitchell in the Journal of the American Medical Association.*

Equine Encephalomyelitis*

By H. W. SCHOENING,† V.M.D.
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SINCE the isolation of the specific causative agent of equine encephalomyelitis by Meyer and his co-workers in 1930 in California, the disease has spread to practically all portions of the United States.

GEOGRAPHICAL INCIDENCE OF EQUINE ENCEPHALOMYELITIS

Since 1935, the U. S. bureau of animal industry has sent a questionnaire to the various state livestock sanitary officials and bureau inspectors in charge relating to information on the incidence of the disease in their respective states. In 1935, there were 23,512 cases; in 1936, approximately 4,000 cases were reported; in 1937, 170,000 cases were reported; and, in 1938, the outbreak assumed its largest proportions, 184,662 cases being reported to the bureau. During 1938, 39 states were involved—eleven in the East and the other 28 west of the Appalachians. The general incidence for the 39 states involved was 23.5 cases per 1,000 animals, with a mortality of 21.4 per cent. In the eleven eastern states, the incidence was 3.9 cases per 1,000 animals, with a mortality of 90.2 per cent. The incidence in the 28 states west of the Appalachians was 24.8 cases per 1,000 animals, with a mortality of 19.8 per cent.

GEOGRAPHICAL DISTRIBUTION OF WESTERN AND EASTERN TYPES

During the various outbreaks a number of strains of the virus have been isolated and typed. It has been found that the virus recovered from the states along the Atlantic seaboard is of the eastern type, and that the virus recovered from states west of the Appalachians is of the western type. Eastern type virus has been proved definitely in Massachusetts, New Jersey, Delaware, Maryland, Virginia, North Carolina, South Carolina, Georgia, and Florida. Western type virus has been identified in California, Montana, Idaho, Utah, Nevada, Texas, Kentucky, Minnesota, Iowa, North Dakota, South Dakota, Kansas, and Illinois.

Virus has been recovered but not identified as to type in the following states: Colorado, Louisiana, Nebraska, Ohio, Indiana, and Wisconsin. The disease has been diagnosed by clinical and pathological means in Connecticut, Michigan, Oklahoma, New Mexico, and Arizona. The disease has been diagnosed clinically in Maine, Rhode Island, Missouri, Mississippi, Arkansas, Wyoming, Washington, and Oregon.

In 1938, the eastern type virus was identified for the first time from Massachusetts and Florida, and the western type was recovered from two new states, Illinois and Kentucky.

Fifteen strains of virus were recovered in 1938 from the following states: Kentucky, Minnesota, Kansas, Illinois, North Carolina, Florida, and Massachusetts. It was again found that the strains of virus from the states west of the Appalachians were strictly western type and that those from states bordering the Atlantic coast were true eastern type. There was no mingling of viruses. It was also found that commercial chick vaccines afforded complete protection against the 15 strains isolated when tested experimentally in guinea pigs.

The disease was reported in Florida in January of 1939 and the specific virus, eastern type, was isolated in this instance. The weather at this time was mild and there was an abundance of insect life. Since that time, scattered cases of encephalomyelitis have been reported in North Dakota, South Dakota, Iowa, Illinois, Missouri, Oklahoma, Texas, Kentucky, Louisiana, and Florida. But, at this writing, nothing of an alarming nature has been reported. Probably a number of the early cases reported as encephalomyelitis may have been due to other causes.

*Presented at the 76th annual meeting of the A. V. M. A., Memphis, Tenn., August 28-September 1, 1939.

†Chief of the pathological division, U. S. bureau of animal industry.

Preseason vaccination now completed has been practiced to a large extent in territories where the disease appeared last year. The appearance of the disease undoubtedly will stimulate later vaccination.

The veterinary corps of the Army has vaccinated all the horses in the Army, National Guard and R. O. T. C. units with vaccine prepared by the veterinary laboratory division of the Army Veterinary School. About 35,000 horses and mules were vaccinated between May 1 and June 15 with no unfavorable results or disturbing reactions.

Efficacy of Chick-Embryo Vaccine

Additional information on the efficacy of the chick-embryo tissue vaccine has become available since the introduction of the product last year. Continued favorable experimental results have been obtained on the use of the product, both in guinea pigs and in horses. It has been found repeatedly that guinea pigs can be regularly protected by two injections at seven-day intervals of 0.5-cc. doses of vaccine against a severe intracerebral inoculation of the virus ten days after the last injection. In many instances a single dose of 0.1 cc. will afford protection against intracerebral exposure. Limited experiments in guinea pigs indicate a substantial immunity nine months after vaccination. However, the immunity engendered by injections of eastern type vaccine has not been as complete after such a period as that with the western type vaccine.

Immunity Tests

Experimental results in horses still indicate a high degree of protection in vaccinated animals. The bureau recently completed a test of immunity after one year in horses. While some of this work has been reported previously, it seems expedient to review briefly this experiment.

Nine horses were given at a seven-day interval two doses of 10 cc. each of a commercially prepared vaccine "A," western type. Three of these horses were exposed to the virus by intracerebral inoculation three months after completion of the treatment; three horses in six months; and three horses in twelve months. Of the three horses vaccinated three months prior to exposure, two survived and one died of equine encephalomyelitis. Of the three animals vaccinated six months prior to exposure, all survived. Of the three horses vaccinated 12 months prior to exposure, two survived and the third developed typical encephalomyelitis and died. With each lot of vaccinated horses, three control horses were exposed to the same virus in an identical manner. In all instances, all of the control horses promptly developed the disease and died. These results indicate that even one year after vaccination a high degree of immunity was present, since two out of three animals survived heavy exposure.

Duration of Virus Potency

The question of the keeping qualities of the vaccine is an important point. The expiration date allotted to the commercial vaccines is one of six-month duration. Experiments on guinea pigs indicate that a potent vaccine, if kept at ice-box temperature, retains a high degree of potency for at least six months. In one experiment two horses were given two injections of a commercial vaccine nine months after its preparation and these animals were completely resistant when exposed one month later to an intracerebral inoculation of the virus, which promptly produced death in the control animals. Experiments on guinea pigs indicate that the vaccine is not as sensitive to temperature changes as it was first thought to be, since in several instances we had vaccines held for one month at room temperature which showed marked protective properties. With other vaccines, however, some loss of potency was noted when allowed to stand for this length of time. It is undoubtedly a very good practice to keep the vaccines at a low temperature at all times, since the available information indicates that the maximum potency is maintained under such conditions.

Government Supervision of Vaccine Production

Last year the chick-embryo vaccine was prepared by one commercial firm only. The bureau has paid particular attention to the licensing of firms for the preparation of the chick-embryo vaccine this year, and every

effort has been made by the bureau, with the full coöperation of the various firms, to see that only highly potent vaccine is marketed.

In this plan of supervision, the bureau has tested over 300 serial lots of vaccines prepared by the various commercial firms, representing about 20 million cc. of vaccine. In carrying out these tests, the bureau has used over 5,000 guinea pigs. Licenses for the production of the product were not issued to commercial houses until it was determined that they were consistently capable of producing a high class product. It was very gratifying to note the splendid coöperation on the part of the commercial houses, and every effort has been made to see that the products marketed are of proper standard. In addition to tests for potency of the vaccines, tests were made for sterility and for the presence of active virus in every lot of vaccine. Apart from tests made by the bureau, the firms themselves are required to make potency, sterility, and safety tests of every lot of the product before marketing. The viruses used by the firms in the production of the vaccines were furnished in the majority of instances by the bureau. At varying intervals the firms have submitted samples of their virus to the bureau for typing tests in order that a check might be made to see that there has been no mixture of the eastern and western types.

CAUSES OF REACTIONS FOLLOWING VACCINATION

As previously stated, a large amount of the vaccine already has been used in the field prior to the advent of the disease. There have been some reports on reactions in horses following the administration of the vaccine. While these have been very few in number as compared with the large number of horses that have been vaccinated, they have caused some concern. The reactions discussed here are those which are considered of a serious nature and do not include ordinary slight swellings at the site of inoculation, which in some animals persist for several weeks but are of no particular concern. The reactions considered have been reported to vary from a severe local swelling without any general disturbance in the animal's health to a severe swelling with systemic disturbance and, in some cases, followed by death.

A study of the situation indicates at this time that the vaccines themselves apparently are not responsible for these reactions. Rigid sterility tests conducted by the bureau and the commercial firms have failed to show any evidence of contamination in the product. Since the product is prepared by the addition of 0.4 per cent formalin, some extensive tests were conducted by the bureau on a number of commercial vaccines, in which a small amount of vaccine was inoculated into large amounts of various types of media, both aerobic and anaerobic. These were incubated for several months, and all cultures remained sterile. It also has been reported and observed that the same serial lot of vaccine that had produced a reaction in an animal or animals was also used on a large number of animals with no unfavorable results. The exact cause of all of these reactions can not be given at this time. However, they could be explained as follows:

First, the technic of administering the vaccine may be faulty. It is possible that pathogenic contaminating organisms on the skin of the animal, on the needle, or in the syringe may be introduced under the skin with the vaccine. It is possible that horses kept under various conditions may harbor on their skin a wide variety of microörganisms, some of which may be of the anaerobic type, and unless good technic is employed in thoroughly sterilizing the skin preparatory to the injection of the vaccine, these might be introduced into the animal in the act of vaccination. This might be of greater moment if the vaccines are given intramuscularly.

Since the vaccine contains 0.4 per cent formalin, which is somewhat irritating to the tissues, it is possible that any contaminants introduced into such irritated tissue might find a more favorable medium for growth than with other types of subcutaneous inoculation. Undoubtedly, many of the reactions that have occurred might be attributed to this point. Therefore, it is of the greatest importance that careful technic

be employed in the administration of the product. This would include not only the shaving of the area, thorough cleansing with alcohol or some other disinfectant, drying, and painting with iodine, but also the use of sterile needles and syringes.

It is also believed that the injections should be made slowly and, preferably, in two or three places in order not to cause undue injury or destroy tissue cells. This can be done either by injecting in separate areas or by partly withdrawing the needle from the original site, turning the course of the needle and injecting the vaccine into two or three places in this manner. After the needle is withdrawn, the area should be painted with tincture of iodine. The second injection should not be made in the same area in which the first injection was made. There is a possibility that the reactions might be due to a sensitization of the tissues by the first injection. This is the second point that might account for reactions.

The third possibility of the cause of reactions might be the fact that some animals are sensitive to chick protein or foreign proteins in general. While many of the reactions reported occur following the second injection, a number occur with the first injection, and while the information we have at the present time indicates that most of the reactions occur following the second injection, this may not represent the true situation, since a further study might not bear this out.

In an effort to determine the cause of the reactions, we have inoculated a number of animals repeatedly with various vaccines in various ways and at various times. We have failed to produce any of these reactions, except in two cases. In one case an animal had been given four injections of the vaccine. Two injections a week apart were given, with no reactions occurring either time. Some months later the animal was given two more injections a week apart. Following the second injection of the last treatment, a large swelling, hard and tense, was observed on the side of the neck from the throat latch to the sternum about 18 hours after the injection. The animal was bright and was not off feed or otherwise inconvenienced. The temperature was normal. The following day the swelling had receded considerably and disappeared within the ensuing week or ten days. This animal had also given a reaction to the tuberculin test a year previous. The reaction in the second animal appeared following the first injection of vaccine. The swelling appeared in 24 hours and covered the whole side of the neck. It was hard and tense. The animal was off feed and showed an elevation in temperature. Hot Epsom salt solution was applied immediately to the swelling and there was a noticeable reduction in its size the next day. The treatment was continued on this day and the animal's temperature gradually came back to normal with improvement in appetite. Very careful technic in the administration of the vaccine was used in this case. The vaccine was sterile and other horses treated with the same serial showed no reaction whatsoever.

Effect of Contact with Chickens in the Sensitization of Horses to Encephalomyelitis

In order to determine if contact with chickens would sensitize horses the following experiment was undertaken. Two normal horses were placed in a large field pen with 20 adult chickens. The chickens had free access to all parts of the stable. They were in the manger and otherwise very closely associated with the horses for 30 days. At the end of this time, the two horses were vaccinated in the usual manner with two doses seven days apart. No reactions whatever were noted in these two animals.

Conclusions

From a general standpoint, it is believed highly important that the method of administration of the vaccine should be beyond the pale of criticism. Thorough preparation of the site of the inoculation, the use of sterile needles and syringes, and the administration of each dose of vaccine in widely separated areas are essential in

(Continued on next page)

Studies on Hog-Cholera Virus*

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SWINE have always been considered the only animals susceptible to hog cholera. Possibly for this reason, it has not been thoroughly investigated whether other animals or birds may play a part in the epizoötiology of the disease.

Early investigators[1] in this country demonstrated that hog-cholera virus can be transmitted experimentally through the peccary, goat, and rabbit. The virus did not produce any apparent manifestations of clinical infection.

In Europe, Nicolle and Balozet[2] showed that the virus can be transmitted to man without producing any evidence of disease. Le Chuiton, Mistral, and Dubreuil[3] indicated that the virus became infectious to guinea pigs when it was injected either intratesticularly or intracerebrally. The virus treated in this manner was no longer pathogenic for swine and did not produce any immunity in them. More recently, Jacotot,[4] working at the Pasteur Institute of Nhatrang, demonstrated that hog-cholera virus could be transmitted for several passages through sheep and goats. According to his report, the animals could be infected either by direct contact with sick pigs or by subcutaneous, intratesticular, or subdural injections. Most of the animals showed inapparent infection, but some did show slight changes in temperature and reactions. He stated that possibly the virus multiplied in the sheep and goats.

In the reports cited above it was demonstrated that hog-cholera virus may produce inapparent infection in several animals. Investigations were not made, however, as to whether or not sheep, horses, cattle, etc. play any rôle in the spread of the disease in swine. Such information may be of special value in the control of the disease under natural conditions. Accordingly, a study was undertaken to determine if sheep or calves play a part in the epizoötiology of hog cholera.

METHODS AND MATERIALS

Animals.—The animals selected for these experiments were all in good health and flesh. The sheep were full grown.

Pigs each weighing about 60 pounds were used. They were of a mixed breed and were known to be susceptible to hog cholera. The calves were young scrub heifers and each weighed about 500 pounds.

Source of Virus.—Commercial virus, the type employed for simultaneous swine vaccination, was used. In order to insure virulence of the virus, it was passed freshly through pigs.

Method.—Every possible effort was exercised to safeguard the animals, particularly the pigs, from cross infection. The pigs were kept in individual hog houses and cared for by a person who did not come in contact with other infected animals. The size of each hog house was about 3½ x 5½ x 5 feet. The lower 3 feet was of solid wood construction and was made waterproof. The floor was raised 15 inches from the ground and contained a drain for collecting the urine. The roof was pitched and the door, about 1½ x 2 feet, was placed on the upper part of the higher side. For ventilation the door and windows were covered with heavy wire mesh on the inside and with fine copper screen on the outside. The sheep were isolated by keeping each

*These experiments were conducted by the author at the biological research laboratory, Pitman-Moore Co., Zionsville, Ind., while he was director of that laboratory.

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(*Continued from preceding page*)

keeping untoward reactions at a minimum. Vaccination is a procedure requiring professional knowledge and skill and technic, all of which the veterinarian possesses and should use at all times.

in an individual pen or by tying each to a post in a large barn.

The animals were kept on a balanced feed. The temperatures, total leukocyte counts and the condition of the animals were recorded daily. In the case of the sheep, each was bled on the 7th, 14th, and 21st day following exposure to the virus. The blood from all the bleedings was defibrinated mechanically, adjusted to 0.5 per cent phenol and stored under refrigeration. The sterility of the blood and tissues was determined by the usual cultural methods.

Experimental Procedure—Part I

TRANSMISSION OF HOG CHOLERA VIRUS FROM SHEEP TO SHEEP, FROM SHEEP TO PIG, AND FROM PIG TO SHEEP BY COHABITATION

Sheep 1747 was injected intravenously with 2 cc. of hog-cholera-blood virus that had been freshly passed through a susceptible pig. Three days later sheep 1743 and 1746 were placed in the pen with the first sheep. In order to insure close contact a small pen was selected and the animals were fed and watered from a common trough. Seven days after they had been penned with sheep 1747, they were removed to another similar pen. Care was taken not to carry any of the bedding over to the second pen. At the same time pig 314 was placed in the pen with the two sheep. The animals were watered and fed as stated. Eight days after they had been penned together the pig showed a temperature of 106° F. and a total W.B.C. of 5,400. The pig continued to show typical symptoms of hog cholera and died eight days later. Throughout the experiment the sheep did not show any signs of illness and their temperatures and white blood counts remained normal.

Upon autopsy the pig showed congested lymph glands and the presence of petechiae on the surface of the kidneys and bladder, but otherwise the viscera were clean. Cultures of the heart blood, lung, spleen, liver, and lymph glands were negative. In addition to the typical hog-cholera findings in pig 314, the virus was identified by neutralization tests with commercial hog-cholera-immune serum. Pig 325 was simultaneously injected with 5 cc. of blood taken from pig 314 and 25 cc. of serum. The pig did not develop the disease and was released 22 days after vaccination. Later, pig 325 was injected with 1 cc. of virulent hog-cholera virus, and proved to be immune.

These findings, without a doubt, show that the pig died of hog cholera and indicate that hog-cholera virus may be transmitted from sheep to sheep and from sheep to pig by cohabitation.

The experiment was reversed by injecting pig 272 subcutaneously with 2 cc. of the virus that had been used to inoculate sheep 1747. Three days later sheep 1727 and 1752 were placed in the same pen. The pig developed typical hog cholera and died twelve days following inoculation. Seven days after the animals had been penned together, the sheep were transferred to another pen with pig 323. The animals were kept together for 25 days, but none of them developed any signs of sickness, and were released.

Infection by direct contact is always subject to chance. In view of this, it was decided to determine if sheep 1727 and 1752 had become infected with virus by cohabitation with pig 272. Two cc. of a combined sample of the blood taken from these two sheep on the seventh day after exposure was injected subcutaneously into pig 326. The pig developed typical hog cholera and died 14 days following injection. The autopsy findings were typical of hog cholera, and the cultures of the vital organs and lymph glands were negative. Pig 323 was assumed to be susceptible to hog cholera because it was a litter mate of pig 326. This indicates that possibly pig 323 escaped infection as a result of the chance involved.

The results of these experiments demonstrated that when sheep were exposed by cohabitation either with a sick pig or a virus-injected sheep, they became inapparently infected with hog-cholera virus. In the first part of the experiment pig 314 developed hog cholera as a result of co-

habitation with the infected sheep, proving that symptomless infected sheep may transmit hog cholera to swine. The second part of the experiment showed that a sheep may become infected with the virus as a result of being in contact with a sick pig.

Part 2

PASSAGE OF HOG-CHOLERA VIRUS THROUGH SHEEP

Sheep 1264 was injected intravenously with 2 cc. of hog-cholera-virus blood which had been recently passed through a susceptible pig. The sheep was kept under isolation and its temperature, total leukocyte count and general condition were determined daily. The animal did not develop any clinical manifestations of infection and seven days following the inoculation 100 cc. of blood was drawn from it.

Two cc. of this blood was injected subcutaneously into pig 269. Five days later the pig showed a temperature of 106° F. and its total leukocyte count was 7,000. The animal developed a typical clinical picture of hog cholera and eleven days after inoculation was bled out while in a morbid condition. The autopsy findings showed congested lymph glands and numerous petechiae on the surface of the bladder and kidneys. Outside of these manifestations, the viscera were clean. The cultures of the heart blood, lungs, spleen, liver, and lymph glands were negative.

The above results indicated that the virus had been transmitted through the sheep, and at this point it was decided to determine whether the passage resulted from persistence of the virus or from actual multiplication of the virus.

Sheep 1166 was injected intravenously with 2 cc. of the blood taken from sheep 1264 on the seventh day following injection. The animal was cared for and observed as described above. As in the case of sheep 1264, it did not develop any signs of sickness. Seven days after inoculation it was bled, and the blood was tested for virus infectivity by inoculating pig 270 with 2 cc. The pig developed typical hog cholera and died on the twelfth day after injection. The autopsy and cultural findings were essentially the same as those in pig 269.

In addition to the test on pig 270, the blood of sheep 1166 was diluted 1:100,000 with normal saline and 1 cc. of this dilution was injected subcutaneously into pig 305. The pig developed typical symptoms of hog cholera and died nine days after inoculation. The autopsy findings were typical of hog cholera and the cultures of the organs were negative.

Attempts were not made routinely to identify the virus either from the sheep or pigs by neutralization tests, because the clinical symptoms and autopsy findings shown by the pigs were considered sufficient evidence of their having died of hog-cholera infection.

An average sheep contains about 4 liters of blood. Calculating on this basis, the amount of virus that pig 305 received would be 1 cc. of a 2×10^{-15} dilution. As far as is known, the highest M. L. D. for hog-cholera-virus blood ever reported was 1 cc. of a 1:200,000 dilution.[5] In view of these results, it seems quite apparent that the hog-cholera virus, when injected intravenously, multiplied in the sheep.

At this point it was decided to transmit the virus consecutively through eight additional sheep before studying its properties. The sheep were kept in isolation, cared for, and observed as described. Each was bled on the 7th, 14th, and 21st days following virus inoculation. In each case, 2 cc. of the blood taken on the 7th day was used for the next passage. The blood from the remaining bleedings was stored for further studies.

The virus blood obtained from sheep 1742, 10th passage, was diluted with normal saline 100,000 times, and 1 cc. of this dilution was inoculated subcutaneously into pig 316. The pig developed typical hog cholera and died eleven days following injection. The autopsy findings were those of hog cholera and the cultures of the organs were negative.

As a result of this test, it was decided to transmit the virus through a few additional sheep. Upon testing the blood from

TABLE I—Sample protocol.

Experiment No. 585. Sheep 2, 3, and 4. *Chart No. 1 S*

SHEEP NO.:	1166				1725				1726			
INOCULUM:	PASSAGE 2 BLOOD FROM SHEEP 1265				PASSAGE 3 BLOOD FROM SHEEP 1166				PASSAGE 4 BLOOD FROM SHEEP 1725			
DOSE CC.:	2				2				2			
HOW GIVEN:	INTRAVENOUS				INTRAVENOUS				INTRAVENOUS			
DAY	DATE	T	S	W.B.C.	DATE	T	S	W.B.C.	DATE	T	S	W.B.C.
1	9–24–37	101.6	None	7150	10–17–37	101.0	None	10100	10–26–37	101.8	None	11500
2	9–25–37	102.0	None	9250	10–18–37	101.0	None	8450	10–27–37	101.6	None	5350
3	9–26–37	102.4	None	.....	10–19–37	101.8	None	5000	10–28–37	101.8	None	6450
4	9–27–37	101.6	None	7450	10–20–37	102.0	None	4750	10–29–37	101.6	None	.....
5	9–28–37	102.0	None	6250	10–21–37	101.6	None	5700	10–30–37	101.6	None	5600
6	9–29–37	101.4	None	6650	10–22–37	102.2	None	6450	10–31–37	102.0	None	6000
7	9–30–37	101.4	None	7500	10–23–37	102.0	None	5550	11– 1–37	101.4	None	7200
8	10– 1–37	102.0	Bled	7000	10–24–37	101.0	Bled	6500	11– 2–37	101.0	Bled	6700
9	10– 2–37	101.6	None	6050	10–25–37	102.0	None	5000	11– 3–37	101.4	None	8750
10	10– 3–37	.....	None	.....	10–26–37	101.0	None	.....	11– 4–37	101.0	None	6150
11	10– 4–37	102.0	None	6750	10–27–37	101.2	None	5450	11– 5–37	101.4	None	5500
12	10– 5–37	.....	None	.....	10–28–37	102.0	None	7000	11– 6–37	101.8	None	6000
13	10– 6–37	101.6	None	7000	10–29–37	101.4	None	8000	11– 7–37	102.0	None	7200
14	10– 7–37	102.0	None	.....	10–30–37	101.0	None	5750	11– 8–37	102.2	None	11500
15	10– 8–37	101.4	Bled	6250	10–31–37	101.4	Bled	7500	11– 9–37	102.3	Bled	10000
16	10– 9–37	101.0	None	7250	11– 1–37	101.6	None	6450	11–10–37	101.4	None	11200
17	10–10–37	102.4	None	8000	11– 2–37	101.4	None	7200	11–11–37	101.2	None	10000
18	10–11–37	102.0	None	8250	11– 3–37	101.2	None	7000	11–12–37	101.2	None	.....
19	10–12–37	101.8	None	7500	11– 4–37	102.0	None	6600	11–13–37	101.0	None	10800
20	10–13–37	102.0	None	7000	11– 5–37	101.8	None	6000	11–14–37	101.2	None	11000
21	10–14–37	101.6	None	8850	11– 6–37	101.4	None	4450	11–15–37	102.0	None	9800
22	10–15–37	101.8	Bled	8025	11– 7–37	102.0	Bled	6800	11–16–37	101.6	Bled	7050
23												
24	Released	10–	15–37		Released	11–7–	37		Released	11–16–	37	

T = Temperature (Fahrenheit).
S = Symptoms.

these animals, it was found that the virus was not recovered from sheep 1748, 11th passage. Two cc. of the blood from this sheep was injected into pig 348. The pig reacted to the injection by showing a typical immunization reaction, characterized by an elevation of temperature and a leukopenia. The temperature was 104° F. on the fourth and fifth days following the injection, after which it assumed a normal level. The total leukocyte count was about 10,000 from the fourth to the eighth day after injection. Thereafter it returned to normal. Fourteen days following this inoculation the pig was tested for immunity by an injection of 2 cc. of a virulent virus. The animal died of typical hog cholera, this indicating that it was not immune.

It would be difficult to state what happened to the virus during the eleventh passage. Apparently, it was in some manner altered in the course of the passage, because upon further investigation it was demonstrated that the virus from the tenth passage remained pathogenic to swine when it was transmitted through additional sheep.

The sheep in these experiments did not develop any clinical manifestations as a result of the virus-blood inoculations. They fed well and the temperatures and leukocyte counts remained normal. A sample protocol

TABLE II—Summary of hog-cholera-virus transmission through sheep.

SHEEP	PASSAGE	DATE OF PASSAGE	DATE OF 1ST BLEEDING	DATE OF 2ND BLEEDING	DATE OF 3RD BLEEDING	CONDITION OF SHEEP	VIRULENCE TESTS
1265	1	9–13–37	9–20	9–27	10–5	Good	Virulent
1166	2	9–24–37	10–1	10–8	10–15	Good	1 cc. 1:100,000
1725	3	10–19–37	10–26	11–2	11–9	Good	Not determined
1726	4	10–26–37	11–2	11–9	11–17	Good	Not determined
1728	5	11– 2–37	11–9	11–17	11–24	Good	Not determined
1729	6	11– 9–37	11–16	11–23	11–30	Good	Not determined
1730	7	11–16–37	11–23	11–30	12–7	Good	Not determined
1738	8	11–23–37	11–30	12–7	12–14	Good	Not determined
1739	9	11–30–37	12–7	12–14	12–22	Good	Not determined
1742	10	12– 8–37	12–15	12–22	12–30	Good	1 cc. 1:100,000
1748	11	1–10–38	1–17	1–24	1–31	Good	Not virulent
1749	12	1–17–38	1–24	1–31	2–7	Good	Not virulent
1750	13	1–24–38	1–31	2–7	2–14	Good	Not virulent
1721	14	1–31–38	2–7	2–14	2–21	Good	Not virulent

showing the daily observation of the sheep is shown in table I. A summary of the hog-cholera-virus passage through the sheep is given in table II.

Attempts were made to produce an anti-hog-cholera serum in sheep by hyperimmunization with either sheep-blood virus or hog-blood virus, but these resulted in failures. However, successful results were obtained by using sheep-blood virus with commercial anti-hog serum for simultaneous vaccination of swine. This indicates that sheep-blood virus is relatively virulent, although it did not produce apparent infection in the sheep. This also serves as additional proof of the identity of the virus after passage through the sheep.

In addition to the above experiments, it was thought that it would be of interest to determine how long the blood of a sheep remains infectious to swine following the inoculation of the sheep with hog-cholera virus.

Sheep 1742 (tenth passage) was bled on the 7th, 14th, 21st, and 27th days following the intravenous inoculation. The blood specimens taken on the 7th, 14th, and 21st days were pathogenic, while the one taken on the 27th day was not. Pigs 316, 285, 318, and 317, respectively, were used to demonstrate the virulence of the virus in the above tests. Pigs 316, 285, and 318 developed typical hog cholera and died, while pig 317 did not show any infection. These results show that when sheep were injected with hog-cholera virus, their blood remained infectious to swine for a period of at least 21 days.

Tests were conducted in order to determine if the blood obtained from sheep 1742 on the 27th day after injection contained any immunizing properties. Fourteen days following the first injection, pig 317 was inoculated with an additional 10 cc. of the same blood. Two weeks later the animal was exposed to a virulent virus by inoculation. It developed a typical case of hog cholera and died. This test indicates that after the blood of the sheep became

noninfectious, it did not show any immunizing value.

Part 3

PASSAGE OF HOG-CHOLERA VIRUS THROUGH A CALF

Calf 1117, a young heifer, was injected intravenously with 5 cc. of virus blood obtained from sheep 1166. The calf did not develop any apparent infection and was bled on the third and 14th day following injection. The blood taken on the third day was tested for pathogenicity by injecting, subcutaneously, 5 cc. into pig 283. The pig developed typical symptoms of hog cholera and was bled out while in a morbid condition. The autopsy findings were positive for hog cholera and cultures of the organs were negative. Five cc. of the blood obtained on the 14th day was injected into pig 273, which did not develop any infection. These results suggest that the virus, when injected into a calf, loses its pathogenicity for swine sooner than in the case of the sheep.

This experiment was limited in scope and served only to show that when hog-cholera virus was injected intravenously into a calf, the blood of the calf was infectious to swine for at least three days following the injection.

Attempts were made to produce anti-hog-cholera serum in calves by hyperimmunization with virulent blood virus from either sheep, calves, or hogs, but the results were not successful.

Discussion and Conclusions.—The results of these experiments show that hog-cholera virus was transmitted to sheep both by intravenous inoculation with the virus and by cohabitation with infected animals. Although the virus produced inapparent infection in the sheep, their blood was pathogenic to swine for at least 21 days following infection. In addition, the infected sheep transmitted the virus to a pig by cohabitation.

Hog-cholera virus was transmitted by intravenous inoculations consecutively through ten sheep. The blood taken from sheep 1748 (eleventh passage) was no longer infectious to pigs. However, this was apparently brought about by this particular passage because blood from the tenth sheep produced hog cholera in swine after further passages through other sheep. In view of the fact that it was possible to transmit hog-cholera virus consecutively through more than ten sheep in such a highly potent form, it is quite evident that the virus multiplied within the sheep.

Under the conditions of these experiments it was not possible to produce anti-hog-cholera serum either in a calf or sheep by hyperimmunization with virulent blood virus from sheep, calves, or hogs. It also was not possible to demonstrate any hog-cholera immunizing properties in the blood of sheep in which the blood became noninfectious to swine following virus inoculation.

Sheep-blood virus was used with commercial anti-hog-cholera serum for simultaneous vaccination of pigs successfully.

The results of these experiments strongly indicate that sheep, and possibly cattle, may become carriers of hog-cholera virus and, in view of this, it is entirely possible that these animals may play an important part in the epizoötiology of hog cholera.

ACKNOWLEDGMENT

The writer wishes to express his sincere thanks and appreciation to Dr. S. H. Regenos of the Pitman-Moore Company for his suggestions and constructive criticism during the course of these experiments.

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[5]Personal communication (1937) with Dr. S. H. Regenos, Zionsville, Ind.

Foot-and-mouth disease caused a grave shortage of meat and milk in European countries during the past two years, although such misfortunes, in the secular press, are always blamed on the politicians.

Effects of Heat on Phenolized Hog-Cholera Virus*

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THE EFFECTS of various temperatures on phenolized hog-cholera virus, such as is customarily prepared for commercial use under license of the U. S. Department of Agriculture, is an important subject and concerns all veterinarians engaged in swine practice, as well as those interested in research work with hog cholera.

It is now understood that hog-cholera virus requires constant refrigeration at a temperature not exceeding 45° F., to safeguard its virulence and thereby obtain the proper reaction in animals vaccinated with the simultaneous administration of anti-hog-cholera serum and hog-cholera virus. Convincing evidence of this necessity is found on page 34 of the report of the chief of the U. S. bureau of animal industry for 1924, from which we quote:

> In an experiment to test the belief that hog-cholera virus after being preserved with phenol may lose its potency if exposed to high temperatures such as occur in summer, samples of virus held for short periods of time at summer temperatures were apparently little affected, while longer periods of time, such as 48 hours at 86° F. and seven days at from 92° to 100° F., exerted a distinct attenuating action. On the whole, the results indicate that hog-cholera virus with 0.5 per cent phenol retains its virulence remarkably well under adverse conditions, but that in the heat of summer, with temperatures ranging from 85° to 100° F. or above, the virus should not be kept at room temperature very long, as it may suffer a serious loss of virulence under such conditions.

With a view of determining more definitely the effects of heat on phenolized virus, particularly the thermal point or points at which it is rendered avirulent or nonviable, certain experiments were designed and conducted with an attempt to correlate them with field conditions under which simultaneous virus is ordinarily used by veterinarians in swine immunization.

Prior to the inauguration of experimental inoculation of test animals with phenolized virus heated at various temperatures and for varying lengths of time, many temperature readings by a recording thermometer were taken in August and September, 1938, within the closed trunk carrying compartments of several automobiles and within the closed seating portions of cars. Other readings have been taken in 1939. It is believed that these data, considered in connection with experimental results later set out, have considerable significance to practicing veterinarians.

The hog-cholera virus used in these experiments was, in all cases, that which is known in virus-serum production as "inoculating virus." Inoculating virus is derived from selected cholera-sick pigs and is customarily used in licensed virus-serum establishments for the inoculation of large numbers of pigs for the production of simultaneous and hyperimmunizing virus. The directly pertinent applicable regulations of the U. S. bureau of animal industry with respect to "inoculating virus" are found in B.A.I. order No. 276, regulation 18, section I, paragraphs 2, 3, 4, 5 and 8. For the purposes of this discussion, phenolized inoculating virus may be regarded as simultaneous virus, such as is customarily used in field immunization in the double treatment for hog cholera.

When referring to phenolized virus, we refer to hog-cholera virus to which phenol has been added to make a 0.5 per cent solution in accordance with regulations of the U. S. bureau of animal industry.

Since the experiments covered a period of several months, representative samples from several different serials of routinely produced inoculating virus were taken from time to time as needed. In most instances the virus used in these experiments had

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‡Superintendent of production, The Corn States Serum Co.

been collected and phenolized for a period of three to four weeks prior to use.

The experimental animals used were generally pigs weighing 50 to 110 pounds, purchased as far as possible from single farms for various groups of experiments and selected after personal observation, the objective being to obtain healthy pigs susceptible to hog cholera. Such pigs were transported in disinfected trucks to segregated quarters on the premises of The Corn States Serum Company, Omaha, Neb., which, operating under U. S. veterinary license No. 99, furnished all hog-cholera "inoculating" virus, together with experimental animals, equipment and facilities, under permission of the chief of the U. S. bureau of animal industry to conduct such experiments on the premises of the licensee in coöperation with the inspection staff* of the bureau assigned to that station. Special sanitary and quarantine measures were observed about the quarters at all times.

The heating of virus was accomplished in some instances by exposure in a bacteriological incubator, the virus samples being placed adjacent to the bulb of a recording thermometer previously checked for accuracy. In other instances, the equipment customarily used for the pasteurization of anti-hog-cholera serum under regulations, with a recording thermometer, was used. Temperature charts from the recording thermometers were obtained in all instances, showing the exact time and degree of heat exposure.

After heating, samples of each virus serial used were cultured. Also, stained smears of the heated virus were examined microscopically for contamination. No growth developed in cultures in any case and the stained smears were free of organisms.

Clinical observations and autopsy findings on experimental animals were made and charted in accordance with the usual standards of the bureau. The coöperation of bureau veterinarians and the technical staff of the licensee company brought together men of extensive experience in actual virus production and inspection in licensed plants and otherwise, accustomed to the daily diagnosis of hog cholera from antemortem and postmortem findings. In addition, leukocyte counts were made in certain instances to substantiate the diagnosis of hog cholera.

Thirty experiments, involving approximately 425 pigs, were conducted. Each experiment was planned to include a sufficient number of pigs to justify the conclusions drawn. For purposes of clarity, pigs receiving heated virus are referred to as "test" animals.

Along with each group of test pigs selected to receive heated virus, there were also selected from the same bunch of pigs at least five animals, designated here as "checks," to receive injections of a representative unaltered sample of the same inoculating virus serial as originally prepared. Also, there were selected from the same bunch of pigs five animals to serve as "controls" for pen infection, which received nothing on the inauguration of the tests.

PHYSICAL CHANGES IN HEATED PHENOLIZED HOG-CHOLERA VIRUS

Incubation or heating of phenolized virus at various high temperatures produced marked changes in the physical properties of the virus. Incubation at 36°-37° C. (96.8°-98.6° F.) for 24 hours produced little if any change of color and a scarcely perceptible increase in viscosity. Incubation at the above temperatures for 48 hours brought about a color change tending to a chocolate hue and a slightly noticeable increase in viscosity; for 72 hours—a distinct chocolate-brown color and a noticeable increase in viscosity readily discernible through the glass container; at 97 hours—a light chocolate color and thick viscid consistency. The virus could be aspirated into syringes with some difficulty and poured with some difficulty. At 120 hours there was little change of color from the preceding 97-hour color, but partial solidification occurred on standing. When the virus was incubated at temperatures and for periods which rendered it avirulent, it was possible

*We are indebted to Dr. E. H. Carter, inspector-in-charge, virus-serum control, U. S. bureau of animal industry, Omaha station, for his close personal collaboration in observing experimental animals and assisting in diagnosis.

to aspirate it into syringes only after severe shaking or agitation of the containers.

When phenolized virus was incubated at 112° F. for four hours, there were slight changes in the color and viscosity. At six hours the color was distinctly brown and the consistency viscid. At eight hours the color became chocolate brown and the consistency thick. At 10 hours, the color was chocolate brown and the consistency semi-solid and pasty.

In virus heated for one hour at 50° C. (122° F.) there was a distinct chocolate-brown color and thick consistency. The color changed and the consistency became more firm as the temperature was increased. When a temperature of 60° C. (140° F.) was maintained for one hour or longer, the virus was solidified and could not be aspirated into a syringe.

EFFECTS ON VIRULENCE OF VARIOUS TEMPERATURES FOR VARYING LENGTHS OF TIME

Phenolized hog-cholera virus heated or incubated at temperatures and for lengths of time stated below possessed sufficient virulence consistently to produce typical or, in a very few instances, atypical hog cholera when injected into susceptible pigs:

122° F. for 1 hour.
131° F. for 1 hour.
137.3° F. for 30 minutes.
96.8°-98.6° F. for 24, 48, 72, 97 and 122 hours.
112° F. for 4, 6, 8 and 10 hours.
On a temperature curve beginning at 104° F.; rising to 120° F. in 3 hours; falling to 86° F. in 6 hours.
On a temperature curve beginning at 115° F.; rising to 130° F. in 2 hours; falling to 100° F. in 4 hours.

Of 137 pigs inoculated with viruses heated at the above temperatures and times, 133 pigs developed hog cholera.

VIRUS RENDERED AVIRULENT

Phenolized hog-cholera virus heated at 58.5° C. (137.3° F.) for one hour did not produce hog cholera in seven inoculated pigs; phenolized virus incubated at 36°-37° C. (96.8°-98.6° F.) for 168 hours produced hog cholera in only one of 17 inoculated pigs; phenolized virus heated at 36°-37° C. for 144 hours produced hog cholera in seven of 30 inoculated pigs, divided into three test groups, in one of which eight pigs developed the disease through association with animals infected from the original inoculation; phenolized virus heated at 36°-37° C. for 156 hours produced hog cholera in five of ten inoculated pigs.

VACCINES PRODUCED BY INCUBATION OF PHENOLIZED HOG-CHOLERA VIRUS

In the conduct of experiments referred to in the preceding paragraph it was demonstrated that the application of heat at the temperatures and for the periods stated produced properties of a vaccine nature in the phenolized virus and its injection into susceptible pigs produced an immunity to hog cholera in a percentage of the test animals inoculated.

Of 43 pigs which survived the original inoculation of heated phenolized viruses, 29 withstood an injection of known virulent virus administered at periods ranging from seven to 14 days after the first inoculation. Twenty-five of the 29 pigs are alive at this date, four having lost identity after being turned into feed lots. These 25 hogs will be subjected at various dates to hyperimmunization as a test of the duration of immunity.

It will be noted that the vaccine properties referred to developed both in cases of long exposure of virus to heat at low temperatures and for shorter periods at much higher temperatures. It was also evident that a certain amount of time was required after inoculation with phenolized hog-cholera vaccine for the development of immunity. The highest percentage of immunity in test animals was demonstrated when exposure to known virulent virus was deferred to 14 days following the original inoculation, while the lowest percentage of immunity was demonstrated in seven days following the original inoculation. It was likewise evident that pigs inoculated with phenolized hog-cholera vaccine were highly susceptible to pen infection in the period immediately following inoculation. In one group of ten pigs inoculated with virus heated for 144 hours at 36°-37° C., two pigs developed symptoms on the fifth and seventh days and all pigs in the test suc-

cumbed to hog cholera from association with these two sick pigs, while in another group of ten pigs inoculated at the same time with the same heated virus, all remained well and later withstood injections of known virulent virus.

The phenomenon of vaccine development in heated phenolized virus is referred to on pages 18 and 19 of the 1935 report of the chief of the U. S. bureau of animal industry, wherein the use of various chemicals, including glycerin, phenol, and crystal violet, as attenuating agents in the production of hog-cholera vaccines is discussed.

VIRUS HEATED AT TEMPERATURES IDENTICAL WITH THOSE RECORDED IN CLOSED CARRYING COMPARTMENTS OF AUTOMOBILES

On September 10, 1938, a recording thermometer placed in the rear of a closed trunk compartment of an automobile exposed to open sunlight recorded the following temperatures: 1:00 p. m.—119° F.; 2:00 p. m.—131° F.; 3:00 p. m.—136° F.; 4:00 p. m.—134° F.; 5:00 p. m.—122° F.; and 6:00 p. m.—103° F.

Phenolized virus heated in a serum pasteurizer on a temperature curve identical with the above failed to produce hog cholera in any of nine susceptible pigs, all of which succumbed to hog cholera when injected 14 days later with known virulent virus, indicating avirulence and non-viability in the heated virus.

The atmospheric temperatures recorded by the U. S. weather bureau for the same hours were: 92°, 94°, 95°, 88°, 87°, and 85° F., respectively.

It is therefore evident that hog-cholera virus may be rendered avirulent when transported in the closed carrying compartment of automobiles for several hours where the normal atmospheric temperatures range from 92° to 95° to 85°.

In two other experiments, where the maximum temperatures within trunks of automobiles were 120° and 130° F., the virus heated at corresponding temperature curves remained virulent. The maximum atmospheric temperatures on the same dates were 87° and 97° F., respectively. The temperatures within the closed compartments of automobiles were found to vary widely, depending on the position of the car with respect to the direction of the sun rays, and other factors. The widest spread noted between atmospheric temperatures and car temperatures was 46° F. It was observed in U. S. weather bureau records that monthly maximum atmospheric temperatures for the area for a five-year period were:

	1934	*1935*	*1936*	*1937*	*1938*
July	110 F.	103	114	100	107
August	110	103	110	103	103
September	89	91	101	100	98

VIRUS RENDERED NONVIABLE

Phenolized virus heated at 60° C. for one hour was rendered nonviable. Susceptible pigs inoculated with such heated material later succumbed to injections of known virulent virus administered 14 days after the first inoculation. This indicated that the vaccine properties were not present in the virus so heated. Like results were obtained from heating virus on a temperature curve beginning at 95° F., rising to 140° F. in three hours, and falling to 131° F. in six hours, such temperatures being recorded in the closed trunk compartment of an automobile exposed to direct sunlight on May 30, 1939.

GENERAL DISCUSSION

No attempt was made in these experiments to study the character of the immunity to hog cholera established by the simultaneous use of heated phenolized virus and anti-hog-cholera serum. McBryde has reported that the simultaneous use of crystal-violet vaccine and serum produced a passive immunity only, derived from the serum used. It may therefore be surmised (although not proved in our experiments) that the simultaneous use of serum, and virus heated under such conditions as to develop distinct vaccine properties therein, would likewise produce a passive immunity only.

The exact range of temperatures and period of heating which produce distinct vaccine properties in phenolized virus is unknown. Also, the character and extent of variations in such vaccine properties is un-

known. Likewise, the effect of various degrees of attenuation, caused by heat, on the ability of such heated virus to confer lasting immunity when used simultaneously with serum is largely undetermined.

In our experiments it was evident that the susceptibility of individual pigs had much to do with the results of using certain heated virus. In certain experiments the heated virus produced hog cholera in some of the test animals, while other pigs in the same group were more resistant and withstood the pathogenicity of the heated virus, developing an immunity to hog cholera as a result of its injection.

Referring again to the data of temperatures in closed carrying compartments of automobiles, one notes that a high of 60° C. (140° F.) was recorded when the atmospheric temperature was 96° F. Since it has been reported by Munce and Hoffman and others that temperatures above 59° C. applied to anti-hog-cholera serum as commercially prepared under license resulted in the development of shock-producing properties in such serum, it is believed that serum transported for a great length of time in closed automobile compartments exposed to direct sunlight, when atmospheric temperatures reach 96° F. or above, may likewise develop such shock-producing properties.

It is a reasonable assumption that the various virus diseases which affect man and animals will come under greater control when the nature and characteristics of the causative factors are more fully known and understood. It is hoped that the experimental work reported herein may add somewhat to the sum total of our knowledge of the characteristics of hog-cholera virus, and in some measure assist in the control of hog cholera, which remains the greatest enemy of swine husbandry.

The heavy hydrogen nucleus is five-trillionth of an inch long and shaped like a football.

The number of hairs on the human head is about 88,000 to 104,000, according to Freidenthal's count, cited by *Hygiea*.

Vitamin E in Sterility*

The department of chemistry of the University of Witwaterstrand, South Africa, coöperating with gynecologists and the faculty of veterinary science at the Onderstepoört laboratory, has announced that the results obtained from vitamin E derived from wheat germ oil have been so satisfactory that further medical and veterinary tests are to be undertaken. An early difficulty has been the tendency of wheat germ oil to decompose, but this has been overcome, the report states, and a sample six months old has proved effective.

Hemolytic Anemia Following Sulfanilamide

Cautious doctors who remain unswayed by every therapeutic whim wondered from the beginning just what genre of damage such a drug as sulfanilamide would inflict on the vital cells of the body while it was making its deadly attack upon the swarming cocci of the humors and tissues. Such physicians were a bit leary about those large doses recommended for acute infections, which time and experience have disapproved under certain circumstances.

Medical literature teems with warnings about the too liberal use of this sensational microbicide. Anemia has been the principal fault recorded. A recent example are the two cases of hemolytic anemia,† characterized by auto-agglutination of the red cells, which were caused by large doses of neoprontosil and sulfanilamide. Compared with the enormous doses sometimes given to animals, the amount that these human patients received was not excessive, and yet repeated transfusions were required to save their lives.

The old law that the cells of the body are more vulnerable to the action of drugs than invading bacteria has not been entirely overcome by the discovery of sulfanilamide.

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*From the London correspondent to the Journal of the American Medical Association, August 5, 1939.

†Journal of the American Medical Association, cxiii, August 5, 1939, pp. 488-489.

Some Histopathological Changes in the Skin of Cattle Infected with Larvae of Hypoderma Lineatum

By S. W. SIMMONS,* Ph.D.

Ames, Iowa

WHEN the larva of *Hypoderma lineatum* (De Vill.) reaches the skin of the back of its host, certain histopathological changes are initiated. Some of these are described and illustrated in the present paper. These changes, aside from their purely pathological and physiological significance, have also a direct economic importance. The injury produced in the hide, the rate of repair of an evacuated cyst, and factors delaying rapidity of healing are other significant points considered.

PROCEDURE

Grubby hides were selected at abattoirs in Des Moines, Iowa, and Chicago, Ill., during the winter and early in the spring of 1936. Blocks of skin containing Hypoderma lesions of unknown age were removed, fixed, sectioned, and stained.

In order to secure data on the rate of healing, a calf was obtained in March of 1937 that had 105 grubs. The back of the animal was closely clipped and each cyst was numbered with an electric tatoo needle. The dates of dropping of the larvae were noted and the animal was slaughtered when dropping was complete. In this manner cysts were obtained which ranged in age from 0 to 23 days after larval migration.

All tissue was fixed in Zenker's fixative. Beechwood creosote was used for clearing the blocks, as sectioning was found to be easier than when xylene was employed for this purpose. Paraffin blocks containing embedded tissue were trimmed and immersed in water for several days prior to their being sectioned, as this greatly facilitated the process. The choice of stains was governed by the structures to be illustrated. Eosin-methylene blue was used in the majority of cases.

All larvae removed and identified were those of *H. lineatum*.

RESULTS

Injury Produced.—The process of skin perforation is probably accomplished mainly through dissolution of the tissue by dermalytic enzymes. Proteases are present in the larvae, and Ono[1] states that a hypodermatotoxin from the intestine of esophageal larvae, when injected into the skin of domestic hares and cattle, causes a local dissolution and perforation of the skin. Furthermore, larvae are situated in the open cysts with their caudal end uppermost. This seems to indicate either that the mouth hooks were not used for piercing the skin, or that the larvae had turned upside down after the act. Knipling[2] has shown that Hypoderma larvae do not molt from the time they enter the host until they perforate the skin and that they retain their same small mouth hooks until this time. Such relatively small and fragile organs would seemingly play little part in perforating the skin. The appearance of the tissues also suggests gradual dissolution rather than mechanical perforation.

An inflammatory reaction follows the progress of the larva and even before penetration is accomplished, a cyst of dense connective tissue, infiltrated with leukocytes, is formed about the specimen. Such cellular infiltration may extend a considerable way into the adjacent tissue and may possibly produce a much weakened hide (fig. 16). The exudate in and about the cyst is strikingly hematogenous, but histogenous products are present, along with considerable débris from the parasite itself. Where cysts are in close proximity, the pathological changes from several individuals may coalesce and greatly reduce the

*Division of insects affecting man and animals, Bureau of Entomology and Plant Quarantine, U. S. Department of Agriculture.

value of the hide for leather production in a relatively large area.

A superficial examination of the hide does not always reveal the total injury, as there may be extensive subdermal abscesses, without larvae, several inches from the cyst. Such a condition is injurious to the hide but is not always detected because of the absence of perforations (fig. 15). Débris-filled pockets, which may remain in the subdermal tissue subsequent to healing, also lower the value of hides for leather (figs. 7 and 14). The relatively unidirectional fibers of healed cyst areas are a source of weakness that lowers the value of hides. Larvae that die prior to penetration of the skin also produce a weakened hide.

General Process and Rate of Repair.—Details of repair in an open hypodermal cyst may vary somewhat, but the fundamental principles are fairly uniform. Epidermal regeneration begins as soon as the grub makes an opening to the outside. This is strikingly evident by the growth of this tissue down the inside walls of the cyst (figs. 1 and 13). Such a process reinforces the cyst walls and thus aids in preventing the spread of infection, if present, to adjacent dermal tissue. Before the grub has dropped, the epidermis may have grown down practically to the bottom of the cyst but, as a rule, it does not connect under the parasite.

A large cystic cavity is present immediately after migration of the larva (fig. 1). This is surrounded by a narrow band of newly formed scar tissue, the upper portion of which is lined with an ingrown epithelium (fig. 13). In a few days after migration the base of the cavity is narrowed by the formation of granulation tissue, and it may be filled with débris (fig. 2). Unlike instances in the healing of some Hypoderma esophageal channels,[3] healing skin lesions are not usually rich in fibrin. Proteolytic ferments liberated from leukocytes in their conversion into pus cells are known to inhibit fibrin formation.

The lower edges of the epidermis grow toward each other and join at their borders or just above them (figs. 3, 4, 5, 10, 11, and 12). Complete union may take place in a week after the larva drops (figs. 5 and 12). At this stage there is an invagination of the epidermis, beneath which may remain considerable inflammation and débris. (See fig. 6.) In about two weeks the epidermis flattens out, leaving an apparently healed skin (fig. 7). Actually, however, pockets of cellular and parasitogenous débris may persist for some time, rendering the hide weak at this point (figs. 7 and 14). The next step in healing is the disappearance, by phagocytosis or autolysis, of the subdermal exudate, usually from dermis to hypodermis, closely followed by a replacement fibrosis (fig. 8). In the healing of an open wound, repair usually is progressive from the base upward. In hypodermal

CAPTIONS TO ILLUSTRATIONS ON OPPOSITE PAGE*

Fig. 1. Hypodermal cyst just after evacuation. The epidermis has grown down almost to the lower level of the dermis. Note abscess in lower right hand corner (approx. x 2½).

Fig. 2. Cyst five days after migration of larva. Borders of epidermis have grown closer together above débris-filled pocket in subdermal tissue (x 3½).

Fig. 3. Another 5-day-old cyst presenting a slightly advanced stage of healing. The epidermis is almost closed above the cyst cavity (x 3½).

Fig. 4. A 6-day-old cyst showing further advance in healing with epidermis joining at its borders (x 3½).

Fig. 5. Epidermal junction complete. Cyst cavity filled with scar tissue; age, 7 days (x 3½).

Fig. 6. More advanced stage of healing than in figure 5. The epidermis is flattening out (x 3¼).

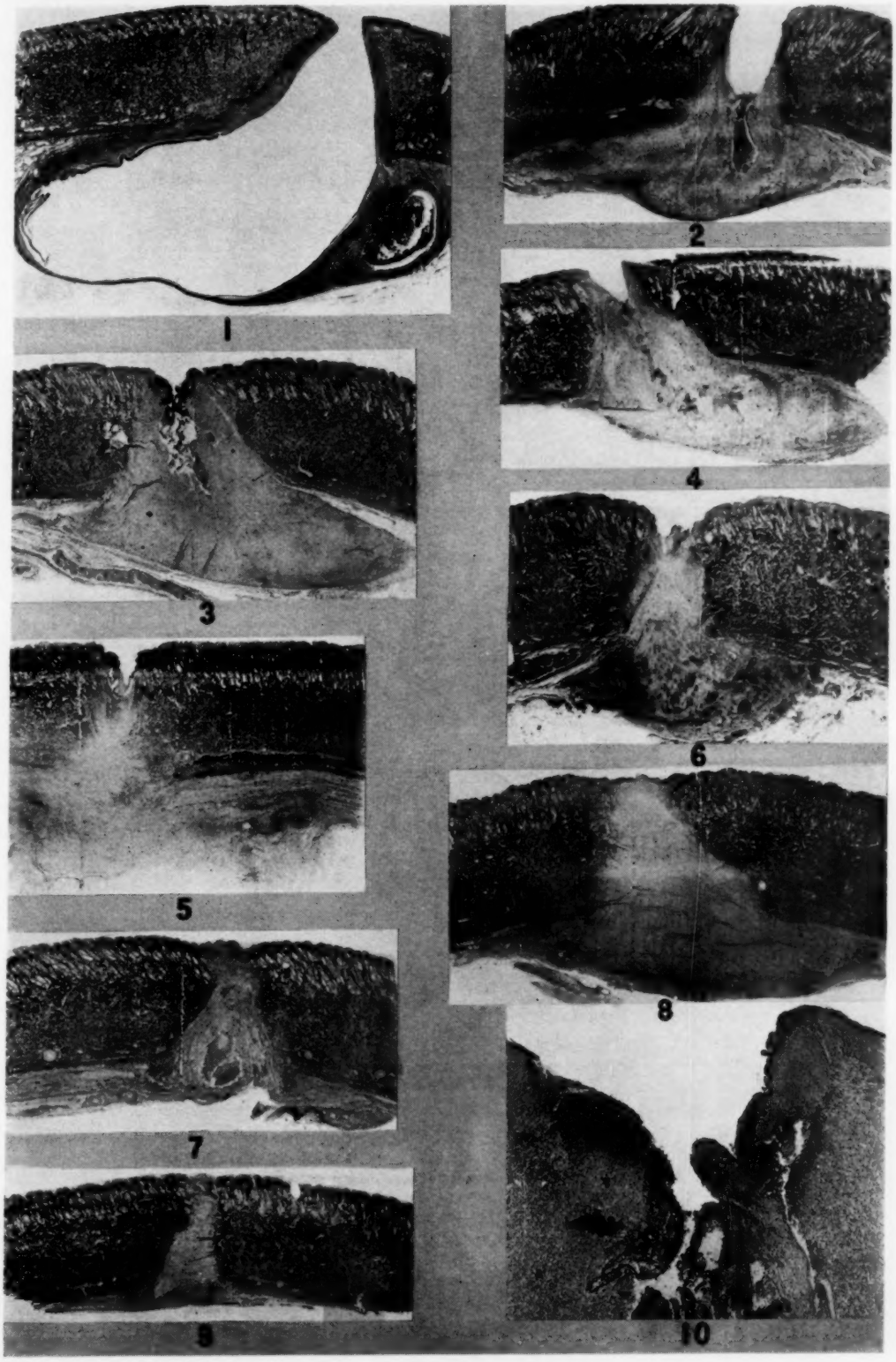
Fig. 7. Complete external healing; age, 16 days. Note débris-filled pocket persisting (x 3½).

Fig. 8. This cyst is only 9 days old but because of the absence of excessive débris it shows more complete healing than figure 7 (x 3½).

Fig. 9. A 23-day-old cyst showing healing practically complete. The scar tissue has contracted to the normal width of the adjacent healthy dermis. Some leukocytes are still present, however, which are demonstrable under high magnification (x 3½).

Fig. 10. Enlarged portion of ingrowing epidermis shown in figure 3 (x 25).

*Most of the figures illustrated are 8 to 10 μ thick and were stained with eosin-methylene blue unless otherwise indicated.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cysts, however, external healing may precede subdermal repair. This is due probably to the interference, at the base of the cavity, by extraneous débris.

After about three weeks the scar tissue has contracted to approximately the normal thickness of the adjacent healthy skin (fig. 9), but even at this time it is usually infiltrated with relatively numerous hematogenous elements, and cicatrization may not be complete. A portion of the parasitogenous débris may be permanently enclosed in the healed area.[4]

Twenty-three-day-old cysts (fig. 9) were the oldest obtained from the animal slaughtered. Therefore, the period required for complete cicatrization in all instances was not determined. It probably averages slightly over a month, depending upon the amount of accumulated débris, especially that of a parasitogenous nature.

The débris in the cyst serves as a support for the proliferating fibroblasts, which gradually replace it. The fibers of the devascularized healed area and of the cyst wall are a dense, uniform mass of collagenous bundles, distinctly marked off from their normal, larger, and more loosely arranged neighbors, of collagenous and elastic fibers (figs. 8, 9, and 13). Early in the healing the papillary layer of the dermis is replaced with fibers more uniformly parallel to the epithelium than those in normal tissue. The reticular layer, although showing slightly more directional differentiation, does not equal in this respect the polydirectional fibers of normal tissue. As pointed out by O'Flaherty and McLaughlin,[4] polydirectional fibers are the source of the tissue's strength, while unidirectional structures are a source of weakness. In this respect a perfectly healed cyst presents a defective hide for leather production.

Cellular Infiltration.—Cellular infiltration is abundant in warble fly infections, whether it be in the skin or the esophagus. As shown in a previous paper,[3] eosinophils are one of the predominating types of cells in esophageal infections. In dermal infections, which are more chronic in nature, the number of eosinophils is relatively much smaller and they are usually confined more to the otherwise unmolested tissue adjacent to hypodermal cysts. Cells of the plasma group usually predominate in the affected areas about the cysts. Certain areas of affected regions are almost exclusively made up of plasmocytes. In figure 16 is illustrated the dense infiltration of this cell about a cyst wall. Foreign-body giant cells are relatively common in and about the cysts.

An interesting phenomenon was the relative scarcity of neutrophils in the tissue about open cysts. Ordinarily such cells are in the majority in most cases of bacterial infection. Their absence was conspicuous in all ages of cysts examined. This may indicate that the bacterial infection is actually less than it grossly appears, or that the

CAPTIONS TO ILLUSTRATIONS ON OPPOSITE PAGE*

Fig. 11. Enlarged portion of joining epidermis shown in figure 4 (x 25).

Fig. 12. Enlarged portion of figure 5, showing complete junction of epidermis (x 25).

Fig. 13. Enlarged portion of figure 1, showing epidermis lining upper portion of cyst wall, newly formed scar tissue, and normal tissue (x 25).

Fig. 14. Enlargement of the débris pocket in figure 7. Note larval cuticle with spines intact (x 25).

Fig. 15. A skin abscess without larva. Focus of infection was larval cyst several inches away (approx. x 2½).

Fig. 16. Cellular infiltration in newly forming scar tissue in healing cyst. The cells are principally plasmocytes (x 375). Delafield's hemotoxylin-eosin stain.†

Fig. 17. A dead larva in subdermal tissue. Such a foreign object may persist for a long while, rendering the hide weak at that point (x 3½). Van Gieson's picrofuchsin stain.

Fig. 18. A larva which died after perforating the skin, and before migration. This greatly retards healing (x 3½). Mallory's anilin blue connective-tissue stain.

*Most of the figures illustrated are 8 to 10 μ thick and were stained with eosin-methylene blue unless otherwise stated.

†The writer extends his thanks to Mr. E. W. Laake for the loan of this slide.

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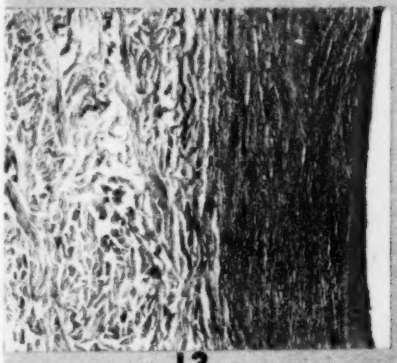

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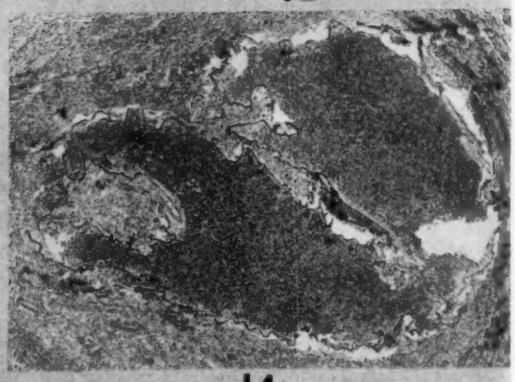

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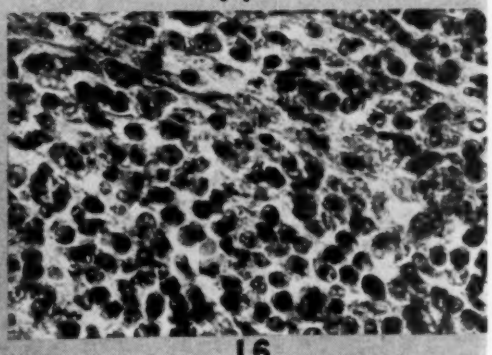

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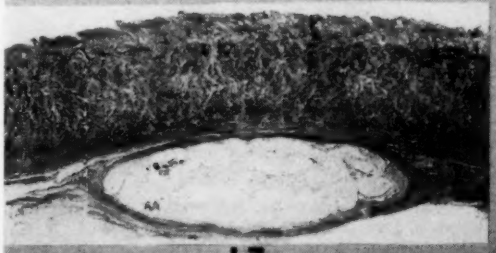

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cells are negatively chemotactic to the secretions of the larvae.

Hyperemia and hemorrhages were rare. A few petechial hemorrhages were found in an occasional specimen.

Effect of Dead, Intact Larvae on Rate of Healing.— Occasionally a specimen is not able to penetrate the skin and dies, at the first attempt, in the hypodermis (fig. 17). This results in a weakened hide that may take months to repair. However, if death results before the dermis is reached, the injury to the hide is minimized. In several instances larvae died while only partially out of the cyst. After the calf was slaughtered, such cysts were sectioned, stained, and studied. It was found that no healing had occurred, even after several weeks, except for a slight ingrowing of the epithelial layer (fig. 18). Cysts from which larvae had dropped normally, several weeks subsequent to the death of those mentioned, showed an advanced stage of healing when the animal was slaughtered.

If grubs are removed from animals four to six weeks prior to slaughter, the hide will have had time for fair repair but where, after the use of insecticides, some of the dead larvae remain in or protrude from the cyst, there will still remain many perforations that will lower the value of the hide for leather. If animals are not soon to be slaughtered, the use of insecticides is probably advisable, as they require much less time to administer than is required by the mechanical removal of the grubs. It is not known, however, that dead grubs left in the skin of cattle to be absorbed do not have some adverse effect on the host. They at least maintain an open port of entry for infection. In practice, however, this has not proved to be of appreciable consequence.

SUMMARY

1. The principal histopathological changes resulting from invasion of the skin of cattle by *Hypoderma lineatum* are described and illustrated.

2. Injury is produced in the hide by: (a) Extensive inflammation initiating a destructive necrosis; (b) accumulation of extraneous débris in the skin; (c) production of larvaless abscesses at points distant from the cysts; (d) the dying and walling off of larvae prior to skin penetration; and (e) regeneration with unidirectional scar tissue fibers, which present a weaker pattern than the polydirectional fibers of normal tissue.

3. Epidermal repair begins as soon as the skin is penetrated, and external healing may be accomplished in two weeks. Complete cicatrization may require three or four weeks, but excessive débris may extend this period.

4. Cellular infiltration is extensive and composed mainly of plasmocytes. Eosinophils and neutrophils are relatively scarce.

5. Larvae may die after penetration of the skin, and before dropping. These may remain in the cyst until absorbed, and thus greatly retard healing. Cysts containing larvae killed with insecticides are thus slower to heal than those from which the specimens have been extracted, or have dropped normally.

ACKNOWLEDGMENT

The writer is indebted to Drs. E. A. Benbrook and H. E. Biester, Division of Veterinary Medicine, Iowa State College, for examination of, and comments on, some of the material used in this study.

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Though phosphorus is a deadly poison, the human body contains about 2 pounds, or 14,000 lethal doses. The body's ways of detoxicating the poisons it contains, and needs, are one of the marvels of biological processes.

Listerella Encephalitis or Encephalomyelitis in Domestic Animals*

By ROBERT GRAHAM,† D.V.M.
Urbana, Ill.

LISTERELLOSIS or Listerella encephalitis and/or encephalomyelitis, a specific bacterial infection of the brain and cord of ruminants, presents to the practitioner a distinct disease entity of the animal encephalitides. The recognition of this disease in cattle and sheep in certain middle western states does not imply a new disease or one which practitioners will recognize as new, but one they have recognized by other designations. The first established outbreak in Illinois (1937-38) was called to our attention in a group of some 200 shipped-in western feeder lambs. In this flock a heavy loss occurred, although prophylactic treatments for hemorrhagic septicemia were administered. Failure to check the progress of the disease, along with other considerations, prompted Dr. L. N. Morin of Clinton, Ill., to request an investigation into the nature of the malady.

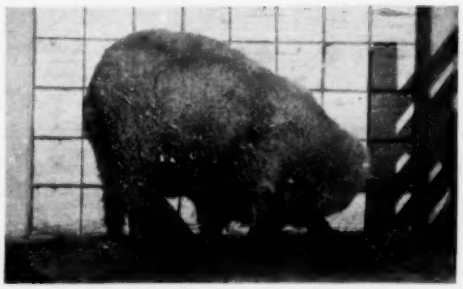

Fig. 1. Ewe affected with listerellosis, field case.

DIAGNOSIS

As a result of the bacteriological and histopathological investigations in this and other outbreaks, only a part of which have been reported, it is apparent that the encephalitides of ruminants may be traceable to bacteriological as well as other factors. Furthermore, it is recognized that the clinician is quite incapable of differentiating the etiological factors of encephalitis and/or encephalomyelitis on the basis of the history and gross pathological changes encountered at autopsy. In the laboratory, however, this is accomplished by establishing the identity of the organism, supported by suggestive, although not pathognomonic, histopathological changes in the encephalon. Thus, the conclusive identification of Listerella encephalitis and/or Listerella encephalomyelitis, heretofore unrecognized, or recognized only occasionally in sheep and cattle by the clinician, requires laboratory assistance.

SUSCEPTIBLE SPECIES

In outbreaks of Listerella encephalitis occurring in Illinois, the causal organism has been isolated from the brain stem only of naturally infected animals. A diagnosis of the disease has been made only upon the identification of the invading organism. As far as is known, no other accurate method of diagnosing this disease or other encephalitides of animals has been employed. While Listerella encephalitis in sheep and cattle has been recognized only recently in the Middle West, and little is known regarding the incidence or distribution of the disease, it is apparent that the malady may have been mistaken for so-called forage poisoning, so-called silage poisoning, gastrointestinal disturbances involving alimentary intoxication, hemorrhagic septicemia, feeding disturbances, acetonemia, or nonspecific cerebro-spinal meningitis, as well as rabies.

It is important, therefore, that the practitioner utilize the laboratory in confirming the existence of Listerella encephalitis in ruminants, and since it is possible that other animals may be susceptible, he should not overlook the possibility of its occurrence in different species. A summary of the susceptible hosts indicates that sheep, cattle, goats, rabbits, gerbilles, foxes, chickens, and man have suffered from spon-

*Presented at the Second Nutrition Conference for Veterinarians, Decatur, Ind., July 12, 1939.

†Division of animal pathology and hygiene, University of Illinois.

taneous infections, while rabbits, guinea pigs, and chickens have proved convenient in establishing pathogenesis in the laboratory diagnosis of this disease.

DISTRIBUTION

Listerellosis is apparently widely distributed through several countries. The first report of the malady was presented by Murray, Webb, and Swann in a description of the disease in rodents in England in 1926. Spontaneously infected rabbits showed an increase in monocytes and they named the causal organism *Bacillus monocytogenes*. The following year (1927) Pirie in South Africa described the disease in a gerbille, a native rodent. The same or an indistinguishable organism was described by this investigator, and it was he who suggested the name Listerella. In 1931, Gill of New Zealand described "circling disease" in sheep and later ascribed it to a Listerella-like organism isolated from the encephalon. Doyle (1932) of Purdue University was probably dealing with the same malady in sheep. At any rate, we have been impressed by the similarity of the microscopic tissue changes described by him in the brain of naturally infected sheep in Indiana to the changes in spontaneous and artificially induced ovine listerellosis.

Jones and Little (1934) in New Jersey described an encephalitis in cattle from which a Listerella-like organism was later identified. During the same year (1934) Burn of Yale University isolated a Listerella from the encephalon in human meningitis. Two years later (1936) this same investigator called attention to the presence of the organism in a prematurely born child, as well as in a child that died eight days after birth. Ten Broeck of New Jersey encountered a spontaneous myocarditis in chickens, while Seastone (1935), in a bacteriological study of the disease, attributed the condition to Listerella. The disease in chickens was characterized by a necrotic myocarditis. Seastone also isolated Listerella from goats. Jungherr of Connecticut (1937), Olafson of New York (1936), and Biester and Schwarte (1939) of Iowa have described the disease in sheep, cattle and sheep, and sheep, respectively, while Cromwell, Sweebe, and Camp (1939) recently reported the isolation of Listerella from the brain of foxes suffering from a distemper-like disease.

In Illinois ovine and bovine Listerella encephalitis have been confirmed in three separate flocks of sheep and four separate herds of cattle. Symptoms of encephalitis were observed in all, yet the clinical diagnosis suggested by experienced clinicians varied from hemorrhagic septicemia to silage poisoning and even rabies, as well as so-called forage poisoning. In addition to the outbreaks heretofore reported in Illinois (1937-38), one unreported outbreak occurred in feeder cattle, which the clinician diagnosed as so-called forage poisoning, and one outbreak occurred in a farm flock of ewes and lambs that was tentatively regarded the responsible agent.

SYMPTOMS

The symptoms of Listerella encephalitis vary somewhat with the different host animals. Sheep appear sluggish and refuse food and water. A common symptom in sheep is the circular movement which caused Gill to use the name "circling disease." The animal keeps its head bent (fig. 1) either to the right or to the left and walks in a circle in that direction. A nasal discharge has been observed, together with conjunctitivis (fig. 2), but these symptoms are not constant. The cornea may be opaque and there is experimental evidence to suggest that a Listerella conjunctivitis may occur independent of the encephalitic syndrome or in conjunction with it.

Animals with brain involvement may lean against a fence or the side of a barn for hours at a time. Finally, weakness and incoördination progress to the point where the affected animal is unable to stand or walk. Death may occur in one to three days to a week or more, after the symptoms first appear. Recovery in mild attacks has been observed. Positive bacteriological findings in the brain of naturally affected animals fortify the clinician in his diagnosis, while an examination of silage being fed in one outbreak which occurred in sheep

failed to yield evidence of the presence of the organism.

It has been observed, however, aside from the neurotropic character of Listerella, that it possesses invasive properties for the conjunctiva of healthy guinea pigs, rabbits, and sheep and may mildly invade the conjunctiva of horses and cattle. Conjunctival instillation of cultures is followed by lacrimation of catarrhal or pustular nature, with clouding of the cornea and an exudative conjunctivitis. The capillaries of the cornea may be noticeably infected. According to Julianelle and Pons (1939) of Washington University, Listerella is the only one of many pathogens that is capable of inducing conjunctivitis following conjunctival instillation.

COURSE

It is not known how the disease is transmitted from one animal to another, but it may be that carrier animals play an important part. The disease does not occur as an explosive type of infection in a herd, nor does it attack every animal. Usually, less than 1 to 10 per cent of the animals may be affected. Several cases may appear simultaneously, while a considerable interval may elapse between cases. The disease in cattle is marked by cerebral symptoms, including incoördination, visual disability, walking into objects, staring and glassy expression of eyes, irritability, emaciation, loss of appetite, and coma, and terminates in decumbency. An animal in a decumbent position may survive for two or three days in a comatose state.

GROSS LESIONS

In chickens the principal lesion of the disease was found on postmortem examinations as a diffuse necrosis of the myocardium. At autopsy examination of sheep and cattle no gross changes have been observed in any of the internal organs. Upon close examination the meninges may show localized, discrete, subacute, inflammatory areas. In rodents a monocytosis is regarded as pathognomonic, but the blood picture noted in these animals has not been observed in ruminants.

The lesions in the brain are microscopic in nature and can be demonstrated in stained sections, although in one proved bovine case the cerebral meninges were highly congested, with a few scattered submeningeal blood extravasations. Oftentimes Listerella may be isolated in pure cultures, confirming the presence of Listerella encephalitis. Other causes of encephalitic symptoms, such as poisonous plants, chemical poisons, hemorrhagic septicemia, acute gastroenteritis, acetonemia or ketosis, rabies, and Aujesky's disease or mad itch, should be excluded in a diagnosis of listerellosis. The occasional development of conjunctivitis in animals showing cerebral symptoms, as well as in apparently normal animals in the herd or flock, may be regarded with suspicion, if not of diagnostic significance.

Fig. 2. Experimentally induced keratitis and conjunctivitis of sheep due to Listerella infection.

PREVENTION OR CONTROL

No specific treatment is known for listerellosis. Isolation of affected animals together with symptomatic treatment may be useful in connection with sanitary measures. There is no evidence to suggest that rations including silage may be contaminated, and in two herds that we observed, a complete change of rations did not noticeably arrest the development of the disease. The fact that closely related, if not identical, strains of Listerella have been isolated from many different species of animals suggests that other animals may possibly be involved in the epizoötiology of the clinical disease in sheep or cattle.

Since Listerella has been encountered in human encephalitis as well as otitis media, and is apparently sometimes fatal in man,

every precaution should be taken by veterinarians in conducting postmortem examinations and especially in removing the brain of Listerella-infected animals. The meat of suspicious cases should not be handled without protective clothing and gloves and should not be passed for human consumption.

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Organized Medicine

The American Medical Association was organized in 1847; the American Veterinary Medical Association in 1863. The former has 113,113 members; the latter 5,370. Both of these organizations have served the people of the United States by advocating and defending a system of professional conduct of the highest order. They erected monuments to their respective labors that no irregular system of medicine has been able to destroy.

A New Way of Keeping Milk

Market milk is an extremely perishable product. Even under the best of ordinary refrigeration it remains potable but for a short time. J. G. Davis of the National Institute of Research in Dairying (England) has described a simple means of increasing the keeping time of milk. The method consists of adjusting the pH of milk to 5.2 with hydrochloric acid and maintaining a temperature of 36° to 38° F. Milk thus treated will keep for two weeks, which is three to four times as long as the commercially cooled pasteurized product. When milk thus preserved is to be used, it is brought back to its original pH with the same amount of dilute sodium hydroxide. The treatment does not change the flavor.

The Gopher as a Transmitter of Encephalomyelitis

The gopher *(Citellus richardsoni)*, or North American ground squirrel, was incriminated as a carrier of the virus of equine encephalomyelitis virus in a series of experiments conducted by Ronald Gwatkin* of the Veterinary Research Station at Lethbridge, Alberta. From a summary of the report we quote:

> A healthy gopher, which harbored engorged nymphs of *Dermacentor andersoni* when captured, was infected with guinea pig brain by the intracerebral route. After death of this gopher, blood was expressed from the ticks and a suspension of this, injected intracerebrally, killed one guinea pig, while another was ill but survived. The survivor resisted a challenge inoculation of guinea pig brain 25 days after the first injection, while the control animal died.

Thus the suspicion that the gopher—a rodent susceptible to the action of the virus in question—with its arachnid parasites is responsible for perpetuating the disease from season to season is not without scientific background.

The part played by rodents and their ticks *(D. andersoni and D. variabilis)* in transmitting Rocky Mountain spotted fever and bubonic plague is no longer debatable.

*Ronald Gwatkin. Canadian Journal of Comparative Medicine, iii (May, 1939), pp. 131-133.

The Control of Animal Diseases in the Philippines

By GREGORIO SAN AGUSTIN,* D.V.M.
Manila, P. I.

ONE OF the most important activities of the bureau of animal industry, entrusted to it by law, is the control of prevailing dangerous communicable animal diseases and the prevention of their introduction into the Philippines through the importation of animals from abroad, this work being assigned to the animal disease control division. The research, investigation and production of the necessary biological products for such diseases are undertaken by the veterinary research division of the bureau.

CONTROL AND ERADICATION OF ANIMAL DISEASES

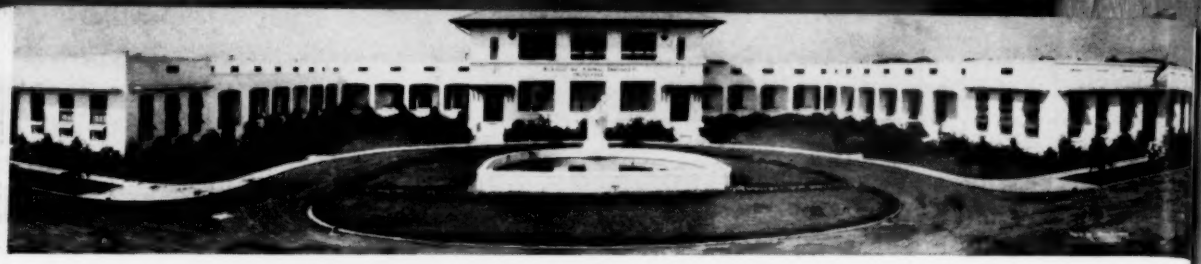
The first effort of the government to carry out this work began in 1886, when rinderpest gained entry into the Philippines, presumably through animals imported from French Indo-China, and caused a heavy toll on cattle and carabaos in different sections of the country. A commission was appointed to study the disease with a view to finding some means of controlling it, but little was accomplished. During the establishment of the American regime, control measures were inaugurated but not until the implantation of the civil government was the work given closer attention. According to the census of 1903, 629,176 head of carabaos and cattle died of the disease in 1901 and 1902.

Creation of the Government Laboratory and the Board of Health.—One of the immediate measures taken by the government to combat the severe epizoötic of rinderpest was the establishment in 1902 of the government laboratories and the board of health. The former provided adequate facilities for the study and submission of scientific reports upon the cause, pathology and methods of diagnosing and combating diseases of man, animals utilized for food, and other domestic animals. The board of health was empowered to enforce regulations for the prevention and suppression of dangerous communicable animal diseases and to decide upon a suitable location for laboratories for the production of vaccine, virus, serum and prophylactics. A serum laboratory was organized and the production of anti-rinderpest serum was started in 1902. A veterinary division also was created for the purpose of investigating and eradicating diseases of animals.

Creation of the Bureau of Agriculture.—In 1901, the Philippine commission enacted a law creating the bureau of agriculture, the functions of which, among other things, were to investigate and disseminate useful information on the animal resources of the Philippines, to determine the practicability of introducing foreign animals, and to develop the breeds of domestic animals found in the Islands. To this new bureau the veterinary division of the board of health was transferred in 1905 and, from then on, the campaign for the control of animal diseases, especially rinderpest, was intensified, the work being entrusted to the division of animal industry.

Establishment of a College of Veterinary Science.—In order that the government might cope adequately with the animal disease situation at that time, it was found necessary to have men trained along modern requirements, and as the services of trained men were not available, the training of local talent seemed essential because outside help proved expensive and not altogether expedient. A college of veterinary science was therefore established in the University of the Philippines by an act of the Philippine legislature. Although the act was passed in 1908, the college was not opened until 1910. Since 1914, when the first graduation was held, this college has been providing the government with practically all of the men it needs, not only for animal disease control work but also for its

*Director of animal industry.



Main building of the bureau of animal industry, Pandacan, Manila.

other activities along animal industry in general.

It is recalled in this connection that the first veterinary school in Europe was founded in France in 1761, precisely for the same reason—the control and possible eradication of rinderpest—that prompted the Philippine government to establish the local veterinary college.

Creation of the Bureau of Animal Industry.—Through the successful control of animal diseases, particularly rinderpest, the production of live stock was encouraged and there arose a need for the organization of a separate entity to devote exclusively to the work of developing the animal industry of the Islands. The bureau of animal industry was therefore created in 1930 by dividing the bureau of agriculture into two distinct bureaus, the other one being the bureau of plant industry. For the administration of the animal work the Islands were divided into 15 veterinary districts and each district placed in charge of a veterinarian.

Present Organization of Animal Disease Control Functions

Rinderpest.—The eradication of rinderpest has always been the main objective of the government. This is now the primary concern of the bureau and, from all appearances, rinderpest will soon be conquered. During the onslaught of this disease in 1900, losses were so heavy that draft and beef animals had to be imported from abroad because agricultural operations in many places were paralyzed. The beef supply in Manila and in some provincial towns was almost depleted. Thereafter it took a heavy yearly toll and the country found it necessary to continue the importation of carabaos and cattle as well as to pass a law prohibiting their slaughter for public consumption.

At that time control measures in the form of rigid quarantine were inaugurated. Later, anti-rinderpest serum was first used both as a prophylactic and as a curative, but with little success. The simultaneous method of immunization in its various modified forms was then tried but was found inadequate as a means of controlling outbreaks of countrywide proportions. It served only to protect individual animals and groups of animals in certain localities and was ineffective as a means of localizing and exterminating the foci of infection.

With rinderpest vaccine prepared by the bureau first used in several injections and, later, in single injections, conferring an immunity of at least two months, the foci of infections were eradicated by rapid mass vaccinations of all carabaos and cattle in the infected and adjoining territories. Through general vaccination carried out simultaneously in the several infected provinces, rinderpest, which was the worst scourge of our carabaos and cattle for almost half a century, has been placed under control. At present, the disease is confined to the southern portion of the Island of Negros.

Anthrax and Hemorrhagic Septicemia.—Anthrax has constantly existed in the Islands but a serious outbreak was not registered until 1922, when the disease appeared in epizoötic form, killing thousands of carabaos and cattle and seriously affecting agricultural operations in some localities. Eighty per cent of the work animals died from the disease. Hemorrhagic septicemia prevails all over the Islands and in certain areas sometimes causes a heavy death toll to carabaos. It was first noted in 1911, although it might have been here long before that time.

The control measure adopted to combat anthrax in 1922 was a seasonal vaccination of carabaos and cattle in and around infected areas, although quarantine was also resorted to as a secondary measure. During that outbreak the Pasteur spore vac-

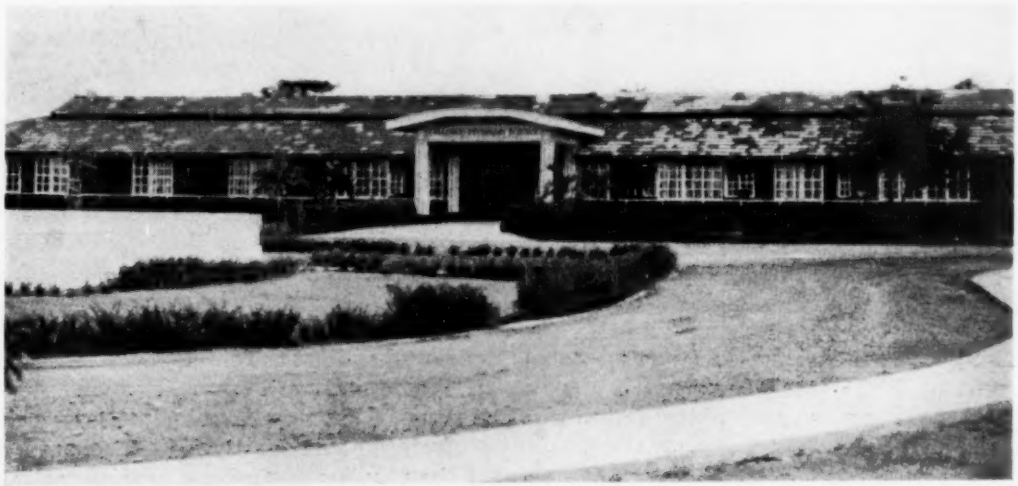
cine was first used, followed the year after (and until 1935) by the simultaneous inoculation of anti-anthrax serum and anthrax spore vaccine imported from the United States. On account of limited appropriations, very little vaccination work was performed. However, since 1936, when anthrax spore vaccine was produced by the bureau at a great saving over the imported vaccine, vaccination has been carried out on a large scale. For hemorrhagic septicemia the control measures first adopted were quarantine and change of pasture but, later, vaccination with imported hemorrhagic septicemia aggressin was inaugurated. Due to a limited appropriation, however, this form of vaccination was used only where the animal owners could afford it. In 1936, the bureau began to manufacture hemorrhagic septicemia vaccine and, since then, vaccination has been done on a large scale.

With the prompt administration of anthrax spore vaccine in outbreaks of anthrax and the use of hemorrhagic septicemia vaccines, together with seasonal mass vaccination in places where these diseases are known to be endemic, losses from the outbreaks have been reduced to the minimum.

Foot-and-Mouth Disease.—This disease gained entrance to the Philippines in 1911, when several infected shipments of live cattle arrived, followed thereafter by a heavy importation of beef, work and breeding animals. Some of the infected animals found their way to several provinces, spreading the disease so rapidly that almost all provinces were infected at one time or another. Although it has disappeared in many places, foot-and-mouth disease has gained a strong foothold in certain localities, especially where the Nellore cattle blood predominates.

As a result of the strict surveillance of all infected regions and the interisland shipment of animals from these places, and the importation of live cattle from abroad, the disease is now confined to definite bounds and its eradication is believed to be in sight. Research work has been instituted by the bureau with a view to discovering some biological products that will break the ring of recurrence which often takes place.

Surra and Glanders.—Surra is prevalent all over the Islands. It was introduced in 1902 and has taken a heavy death toll among the equine species, thereby hindering development of the horse industry in the country. Glanders is also widespead, especially in centers where horses are still used to a great extent for transportation. Microscopic examinations are made of the blood, and quarantine, rigid examinations and, if necessary, serological tests are employed in the case of animals which are taken to surra-free islands, in order to



Main building of the College of Veterinary Science, University of the Philippines, Pandacan, Manila.

check the spread of the disease. The bureau is working on a treatment for surra which is giving satisfactory results. To control glanders, tests are also made and, as in the case of surra, those found positive for the disease are recommended for slaughter or destruction.

Bovine Tuberculosis.—Bovine tuberculosis is not rampant in the Islands, except on dairy farms, where mostly imported animals are in the milk line. Judging by the reports of the different slaughterhouses in the Philippines, the incidence of tuberculosis among native cattle and carabaos is negligible.

The control of this disease is limited to dairy farms, where periodic tests are made of all the animals by the personnel of the bureau. Animals showing a positive reaction are recommended for slaughter or destruction.

Contagious Abortion.—The great interest which certain men have shown of late in the dairy industry has started a move for the importation of dairy cattle from abroad for the purpose of establishing dairies and raising locally dairy cattle of imported breeds. With these importations contagious abortion was introduced into the country.

Restrictions on the importation of cattle have been established in order to minimize, if not entirely to eliminate, the chances of importing affected animals. These measures require imported cattle to undergo contagious abortion tests at one-month intervals at the point of origin. Only negative cattle may be shipped to the Philippines.

Hog Cholera and Swine Plague.—Hog cholera is widely prevalent in most provinces and although statistics on its incidence are meager, we know from observations made in various parts of the Islands that the yearly loss from this disease is considerable. Because of the urgent need in the past of concentrating the activities of the bureau on a limited field, due to a lack of funds to carry on extensive campaigns against other diseases, like hog cholera, this disease has been one of those which could not be given the attention it needed. It did not seem to constitute a public calamity, as rinderpest once was, notwithstanding the fact that the economic losses resulting from it affect the whole country, especially small farmers.

Vaccination against rinderpest.

The most effective control measure now employed against the disease consists mainly of the administration of the simultaneous treatment, but this is limited to large swine projects, which are very few. The majority of the people raising hogs in the rural districts are small farmers who keep only a

few head of hogs, which are allowed to run at large.

The bureau, however, has prepared a biological product for the treatment of hog cholera which is showing good results.

Swine plague, another disease of hogs, is in most instances associated with hog cholera as a secondary infection. The campaign against this disease is therefore conducted in connection with the hog-cholera program. Thus, vaccination against swine plague has been limited to herds that have been given hog-cholera treatment. The swine-plague vaccine, which is also produced by the bureau, is given at the same time as the simultaneous treatment, and separately two weeks later. This method of treatment has given satisfactory results.

Avian Pest, Fowl Cholera and Fowl Pox.—Avian pest is the most destructive disease among fowls in the Philippines. It gained entrance in 1927 and, since then, has spread to many provinces, causing considerable losses to poultry and, in some instances, wiping out entire flocks.

Biological products for fowl cholera are manufactured by the bureau. Roup and fowl-pox vaccines have given satisfactory results when administered on time. For avian pest, however, no vaccine has as yet been discovered, but the bureau's laboratory is constantly investigating the disease with a view to developing a product for its control. Isolation has been found effective as a control measure, provided the healthy fowls are isolated either individually or in groups upon the appearance of the first, second or third case, but not otherwise.

In the field great difficulty has been encountered because the people allow their chickens to run unbounded. Hence, in an outbreak of a poultry disease most of the birds in the flock are exposed before the owners are aware of its presence. Consequently, an intensive educational campaign aimed to instruct the people to keep their chickens in fenced poultry yards is being carried out by the bureau's field men.

Rabies.—Canine rabies is prevalent all over the Islands. Its control has been entrusted by law to the bureau of health but private and government veterinarians coöperate with the health authorities by making diagnoses and performing preventive inoculations against the disease. Whenever cases of rabies are reported to the bureau of animal industry, mass vaccination is employed by its personnel, using the anti-rabies vaccine manufactured by the bureau laboratory.

Bureau of animal industry immunization station.

Other Animal Diseases.—Other prevailing diseases of lesser economic importance are anaplasmosis, Texas fever, and cow pox in cattle; epizoötic lymphangitis, strangles,

and tetanus in horses; swine pox, kidney worms, lung worms, and cysticerosis in swine; nodular disease in goats and sheep; and botulism and blackleg in poultry. These diseases and other ailments that do not fall within the meaning of dangerous communicable animal diseases are handled by the bureau whenever any of them come to the attention of its personnel. Free treatment is given when warranted, especially in places where there are no private veterinary practitioners.

IMPORTATION OF ANIMALS

To guard the country against diseases peculiar to, or prevalent in, other countries, rules and regulations calculated to minimize or eliminate the chances of entry into the Philippines of affected animals have been promulgated by the government. The diseases specified in Animal Industry Administrative Order No. 4-1, entitled, "Rules and Regulations Governing the Importation, Bringing in, or Introduction of Animals into the Philippine Islands from Foreign Countries and Prescribing the Treatment to Which They Shall Be Subjected Before Their Shipment and After Their Arrival," include glanders, surra, dourine, nagana, anthrax, rinderpest, tuberculosis, hemorrhagic septicemia, foot-and-mouth disease, contagious pleuropneumonia, contagious abortion, hog cholera, European fowl pest, fowl cholera, fowl typhoid, and any other acute communicable disease which may cause a mortality of over 5 per cent in one month.

In accordance with the above-mentioned order the importation of animals from other countries is allowed only upon aforegranted permission from the director of animal industry. Animals must be accompanied with a health certificate issued by competent veterinary authorities from the country of origin and, depending upon the diseases prevailing in such country at the time of shipment, a certificate of immunization against, or a certificate of test for, such diseases is also requested. Upon being brought into the Philippines, animals are subject to inspection and, if necessary, placed under quarantine, depending upon their health, or subjected to tests which the director of animal industry may deem necessary.

The importation of cattle is now limited to those intended for breeding, draft, and the manufacture of serum. The importation of animals for slaughter is prohibited by virtue of act No. 3155 of the Philippine legislature, passed in 1924.

Foot-and-Mouth Disease in Germany

At the International Agricultural Congress held in Dresden, Germany, in June, Dr. Müssemier, minister in charge of the German board of health, read a paper on foot-and-mouth disease reviewing the extent of the present epizoötic which has stricken the entire Reich. Up to the end of last year more than 700,000 farms were affected and more than 500,000 head of cattle were vaccinated by the new method signalized at the Zurich International Veterinary Congress in 1938. The International Agricultural Congress was attended by 1,500 delegates from 30 countries.

Dystocia from "Hip Lock" in Cows

A normally presented calf of large dimensions due to breeding immature heifers to Holstein-Friesian bulls may be blocked at the pelvic girdle by its large hips. A straight pull only increases the fixation. The hips resting against the pelvic girdle act as a toggle that tightens with the traction.

The delivery of such a calf may be accomplished, as a rule, by placing the cow in lateral decumbency and drawing the protruding portion of the calf forward over her buttocks. This maneuver is effected by a strong assistant, seated on the cow's body, who pulls heavily upon ropes attached to the calf's fore legs. This sort of traction draws one of the locked hips into the pelvic path and thus breaks the lock. This accomplished, a straight backward pull will easily terminate the delivery. *(J. W. Wadsworth. A Method of Overcoming Hip Lock in Cattle Obstetrics. The Cornell Veterinarian, xxix, April, 1939, p. 144.)*

Roentgen Therapy in the Treatment of Acute Inflammations*

By MYRON THOM, D.V.M.
Pasadena, Calif.

THE USE of roentgen rays as a method of treatment in acute infections and inflammations is far from having the place in veterinary therapy which the results justify. In the opinion of most veterinarians, x-ray therapy is a treatment to be considered only in certain chronic conditions when all other therapeutic measures have failed. This is a serious mistake and is due largely to a lack of knowledge of the benefits obtainable from small doses of x-ray in acute conditions. In the writer's opinion there are but few localized, acute inflammatory or infectious conditions—barring those limited to intestinal tissue—that will not be benefited by suitable doses of roentgen therapy. In many of these conditions x-ray is the therapy of choice and should be used in preference to all other methods of treatment. For example, acute hematoma of the ear promptly disappears with x-ray treatment, leaving no scar or disfiguration. This is very important in valuable show animals.

Regarding the x-ray treatment of infections, one leading American roentgenologist says, "If the results of roentgen therapy in malignancy could in any way compare with the results of only small doses of roentgen rays in the treatment of acute infections and inflammation, there would be relatively little concern about cancer or sarcoma."

Advantages of Roentgen Therapy in Veterinary Practice

We veterinarians who use the roentgen ray in this field are in a better position than the men in human medicine, since we do not have to depend upon referred cases. Medical literature has many references to this problem. One radiologist mentions his excellent results in treating cases of acute tonsillitis but says that most of these patients are physicians or members of their families. In other words, human patients with acute inflammations try home remedies first, then they go to their physician, and after a while they are referred to the radiologist for treatment. By that time the acute phase of the disease has subsided.

It is axiomatic that the more acute the conditions, the better the therapeutic results from x-ray. When a valuable dog develops a hematoma, or acute otitis or similar inflammation, we see it almost at once. The treatment of inflammations by x-ray is a relatively new field, and we are in a better position to utilize it than the radiologist treating human patients. Certainly, the veterinarian can make some valuable contributions to this phase of medicine.

In man the following conditions have been favorably treated with roentgen ray and in many of them it is the present treatment of choice: furuncles, carbuncles, felons, abscesses, erysipelas, gas gangrene, hematoma, conjunctivitis, otitis media, lymphangitis, sinusitis, peritonitis, pneumonia, post-operative wound inflammations and infections, parotitis, and a host of other acute inflammatory conditions.

Stages of Infection Where X-Ray is Most Effective

This valuable adjunct in combating infection should be employed when hyperemia, leukocytic infiltration and other inflammatory phenomena are beginning to appear. At this time the infection itself is not well established and, hence, an added reinforcement of the body's defense mechanism through irradiation therapy may abort the inflammatory process by overwhelming the invading microörganisms.

It is necessary first to review the actual mechanics of the inflammatory process in order to understand the stages at which intervention in the form of roentgen therapy will be most effective. These are:

1. Hyperemia, with dilation of all ves-

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sels and increased rate of blood flow.

2. Temporary stasis or retardation of blood flow caused by changes in endothelia of the vessels.

3. Margination of leukocytes. The white blood cells gradually accumulate along the walls of the vessels as the result of decreased pressure.

4. Emigration of leukocytes. Polymorphs and mononuclears pass through the vessel walls by virtue of their ameboid movement and increased size of intracellular spaces. Shortly after emigration begins, the inflamed tissue areas are densely crowded with specialized phagocytes.

5. Diapedesis of erythrocytes. Extravasation of the red-blood cells follows the leukocytic emigration and the surrounding tissue at this juncture becomes swollen and more fluid than normal. The composition of the blood plasma is altered and the serum or inflammatory exudate now present in the tissues contains a high concentration of fibrinogen.

During this entire process, there occurs a parallel series of tissue changes, during which even more phagocytic cells are produced and deposited in the affected area. It is, of course, after this acute, preparatory stage that tissue degeneration, pus formation, and eventual resolution take place.

BASIC FACTORS DETERMINING THE RÔLE OF ROENTGEN THERAPY

In the foregoing review, there are two basic facts which serve as a working premise in understanding the part played by x-ray in the therapy of inflammations: First, the increase in the number and the emigration of leukocytes to the affected area and, second, the all-important rôle played by the polymorphs as phagocytes.

Lymphocytes.—It is established that lymphocytes are, first of all, body cells in roentgen sensitivity. Radiation produces early nuclear disintegration of lymphocytes and, with their destruction and the resultant relief of pressure, pain is relieved. Beyond the mechanical relief of pain, the disintegration of lymphocytes undoubtedly serves to liberate certain antibodies and enzymes contained within the cells and to render them more available for defense than they are when in the intact cells. Obviously, if the roentgen ray has such a pronounced effect on the lymphocytes of inflammation, the most logical time to employ radiation is at the peak of lymphocytic infiltration—during the most acute stages of the inflammatory process.

Phagocytosis.—In addition to the destruction of lymphocytes, it has been demonstrated that roentgen radiation serves to promote the second important factor in the defense processes, namely, phagocytosis. There is microscopic proof that phagocytosis is most active following radiation and continues for several days. Polymorphs and macrophages are longer lived than lymphocytes under radiation and thus remain in the inflamed area after destruction of the lymphocytes, to continue their important work. It is not unreasonable to presume that the promotion of phagocytosis is in part accompanied by the production of a substance, not unlike opsonin in function, which prepares invading organisms for more rapid ingestion. The ray itself may possibly assume the rôle of a theoretical antigen in stimulating the tissues to the autogenous production of cellular antibodies, whose functions are not yet fully understood.

X-RAY REINFORCES BODY DEFENSE

It is evident that the end result of radiation therapy in inflammations is to change the balance of power in favor of the defense mechanisms of the body, adding reinforcements which assist in overwhelming the invading microörganisms. This is evidenced by the fact that radiation therapy of abscesses, even in the later stages, tends to change the character of the fluid content to a less septic type than the untreated abscess produces. The physiological benefit of this new defense aid is seen in the shortened period of recovery and the diminished toxic reaction which results from the reduction of sepsis. The true explanation of the powers of the roentgen

ray remains an unwritten chapter in the story of radiation therapy. The promotion of autolysis and liquefaction, the production of antibodies and enzymes, and the bactericidal action of the rays are only partially understood today.

EQUIPMENT

The equipment used for this work should have a range of voltage from 60,000 to 140,000. New equipment for human hospital use is so mounted that it may be wheeled from bed to bed, in the manner that radiographic equipment has long been employed. Since the doses used are very small, patients in adjacent beds in the ward are not endangered.

The factors governing this type of treatment are simple and vary only slightly, regardless of the lesion, its stage of development, or its location. The treatment is limited to the part involved; adjacent areas are protected by lead rubber or therapy foil. In acute stages the dose should be 60 to 175 r at a voltage of 60 to 140 kilovolts. The filtration is from 1 mm. of aluminum to 1 mm. of aluminum, plus one-fourth mm. of copper, depending upon the depth of the lesion to be treated. For example, our dose in treating acute moist eczema (eczema rubrum) in dogs is 50 r at 85 KvP and 1 mm. of aluminum filter. In cases involving greater tissue depth, as acute prostatitis, we give 100 r at 140 KvP and through 1 mm. of aluminum plus one-fourth mm. Cu.

All roentgen ray equipment for animal use, either for radiography or therapy, should be shock proof. In addition, the x-ray tube should be enclosed in a lead-steel housing for further safety.

TERMINOLOGY AND DEFINITIONS

It is possible to give as accurate a prescription for roentgen treatment as for any other form of therapeutics. If a radiologist knows the voltage, filter, half-value layer, focal skin distance, and the dose in international r units, any given treatments can be exactly duplicated. For the benefit of veterinarians not familiar with these terms, the following definitions are given.

The international unit of x-ray energy is called an "r" unit. It is defined as that quantity of radiation which, when secondary electrons are fully utilized, produces in 1 cc. of atmospheric air at 0 degrees Centigrade and 76 mm. of Hg. pressure such a degree of conductivity that one electrostatic unit of charge is measured at saturation current. Fortunately, there are r unit meters which will determine this for us under any specified condition.

The filters used with x-ray apparatus are composed of different types of metal. They may be used singly or as a compound filter. The common filters are aluminum, copper and tin. These are inserted between the x-ray tube and the patient. Their purpose is to change the wave length of the x-rays, produced by the tube, and to alter the depth-dose percentage. One-half to 1 mm. of aluminum is largely used for skin work, while copper and tin are used for deep therapy.

Half-value layer, expressed HVL, indicates that thickness of filter required to reduce the intensity (number of r units) 50 per cent. The terms wave length and HVL are important in giving the dose for any lesion.

The anode skin distance (A.S.D.) signifies the distance from the anode of the x-ray tube to the skin. Most skin diseases are treated at an A.S.D. of 30 cm., whereas deep therapy uses 50 cm. in many cases.

The designation KvP means the peak number of volts (expressed in thousands of volts) delivered to the tube.

The term milliampere (Ma) is self-explanatory.

CONDITIONS IN WHICH ROENTGEN THERAPY IS INDICATED

Roentgen therapy is the ideal treatment in the acute stage of the following condition in small animals: Hematoma auris, peracute otitis externa, beginning abscess anywhere in body, parotitis, keratitis, postoperative wound inflammations and infections, and prostatitis.

In the treatment of acute cases of mammitis, peritonitis, pneumonia, metritis, and

sinusitis, roentgen therapy is a valuable adjunct to other forms of therapy.

DOSAGES RECOMMENDED

Hematoma Auris.—In our practice a suitable dosage of roentgen ray is regarded as a specific in acute cases of hematoma auris. In this and all other x-ray treatments we give a dose of morphine or nembutal before treatment. The x-ray dose for hematoma is: KvP 125, F.S.D. 25 cm. Filter 2 mm. al r units 150 HVL 3.8 mm. al. This is repeated the following day. We have rarely had to give more than two treatments for complete absorption of the blood.

Abscesses.—In the treatment of abscesses before they have started to resolve, the following dose is suggested: KvP 125, F.S.D. 25 cm. Filter 3 mm. al r units 90 HVL 4.6 mm. al. This dose is repeated the next day and is usually sufficient to abort the abscess. This treatment is equally applicable in abscess of the cat. If the abscess is nearly ready to break down, radiation acts as a poultice, localizes the lesion, and promotes rapid healing.

Parotitis.—Parotitis and acute inflammation of the adjoining lymph tissue can be readily aborted if the lesion is treated before resolution begins. As in all inflammatory cases, the relief of pain is a constant feature following irradiation. The x-ray dose is: KvP 140 F.S.D. 25 cm. Filter 3 mm. al r units 175, HVL 5.2 mm. al. This dose may be repeated the next day.

Otitis Media.—The cases of peracute otitis media we sometimes see, often in puppies breaking with distemper, are quickly relieved by radiation therapy. One treatment is usually sufficient; if not, treatment may be repeated in five days. The x-ray dose is: KvP 125 F.S.D. 25 cm. Filter 3 mm. al r units 100 HVL 4.6 mm. al.

Tonsillitis. — Acute tonsillitis is best treated under nembutal anesthesia. A mouth gag and tongue forceps are used to expose the tonsils and adjacent tissue. A cone of suitable size attached to the head of the therapy unit is introduced into the mouth. One treatment usually is sufficient. If a second treatment is necessary, it is better to irradiate through the pharyngeal area with higher voltage and heavier filter. The x-ray dose, given through the open mouth, is: KvP 125 F.S.D. 25 cm. Filter 3 mm. al r units 125 HVL 4.6 mm. al.

Keratitis.—Acute keratitis responds well to x-ray treatment. As this condition is usually observed in puppies, considerable care must be taken to avoid an overdose, as the lens of young animals can be damaged by x-ray to the extent of producing cataract. Our dosage for this condition is: KvP 85 F.S.D. 15 cm. Filter 1 mm. al r units 60 HVL 2.0 mm. al. This dose may be repeated the third day if necessary.

Moist Eczema.—The pain attendant to acute moist eczema is greatly relieved and the danger of secondary infection lessened with the following treatment: KvP 100 F.S.D. 25 cm. Filter 2 mm. al r units 50 HVL 3.0 mm. al. This may be repeated for three or four consecutive days, if needed.

TREATMENT OF THOROUGHBREDS

There should be a promising field for this type of x-ray therapy, using portable shock-proof machines, in the treatment of acute inflammations of the feet and legs of race horses. One leading radiologist has stated that any condition that will be benefited by local heat or poulticing can be better treated by x-ray. Portable equipment now available will easily give sufficient depth penetration to reach any part of the leg or foot of a Thoroughbred.

CONCLUSION

The virtues of roentgen therapy in acute inflammatory conditions may be summarized as follows:

1. Pain is relieved rapidly.
2. Fever and toxic symptoms are quickly reduced.
3. The period of treatment is relatively short.
4. Cosmetic results are infinitely superior to radical surgical procedure in conditions such as hematoma, parotitis, and abscess.

(*Continued on next page*)

Is Running Fits a Deficiency Disease?*

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THE DISEASE in dogs known as running fits, fright disease, or canine hysteria[1] has been observed for some time. Frink[1] has noted that sedatives or hypnotics which empty the stomach relieve the condition. He has suggested as the result of practical experience under field conditions that the disease may be dietary in origin.

Studies with growing dogs in connection with another problem indicated that the use of heated proteins in the diet led to disturbances commonly associated with running fits. In order to study the problem more completely we prepared a simplified diet composed of wheat and meat scraps. This diet was subjected to dry heat in a retort kept above 200° C., although the temperature of the product was significantly lower. Dogs fed this ration as the sole article of diet have consistently developed running fits. The evidence presented here indicates that the condition may be alleviated or prevented by the addition to the ration of a sufficient amount of unheated proteins or protein-containing foodstuffs.

EXPERIMENTAL PROCEDURE

The dogs were housed in pens in our animal room. The heated ration was coarsely ground in a Burr mill before use. This ration and the supplements, except where noted, were fed in the dry state. An amount of food more than sufficient to satisfy the daily demand was allotted each morning. Water was supplied in porcelain jars twice daily so that it would be present at all times.

In the instance of dogs 25, 26 and 28, a dry canned dog food supplement was supplied, as will be shown later. This product was a food which was dried in a drying room before fans overnight in accordance with the procedure previously described by the authors.[2] The temperature in the room was never above 60° C. and that of the product was lower. This product was known to be a complete ration, as shown by assays with rats and chicks. The dry canned dog food contained 30 per cent protein while the heated ration contained 22 per cent protein.

Dogs 25 to 28 inclusive were litter mates placed on experiment when 6 weeks old.

RESULTS OF EXPERIMENTS

Dog 22.—This animal was about 2 or 3 months old, predominantly Spitz. It was fed the heated ration as the sole article of diet. The initial weight was 5,800 gm. The dog steadily lost weight and, on the eighth day, had a severe attack of running fits. It appeared to be temporarily blinded, snapped when an approach was made, walked or ran into the sides of the cage, and barked continuously. The weight was 5,450 gm. One deworming pill was administered to rule out worms as a causative agent.

On the ninth day of the experiment the animal continued to show running fits. One hundred gm. of crude casein was placed in a separate container in the cage. All of the casein had been eaten by the following morning and there was marked improve-

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†Wilson & Co. fellow.

(*Continued from preceding page*)

5. Where proper dosages have been given, no deleterious effects have been observed.

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ment in the condition of the animal. The heated ration was supplemented with 5 per cent of crude casein.

Three days later the dog appeared to be in good condition. No attacks of running fits had been noted since the ninth day of the experiment. The weight was 5,600 gm.

On the 18th day the dog was found dead. The result was not wholly surprising, since the heated ration was deficient in other factors. The level of casein was probably insufficient to give complete protection.

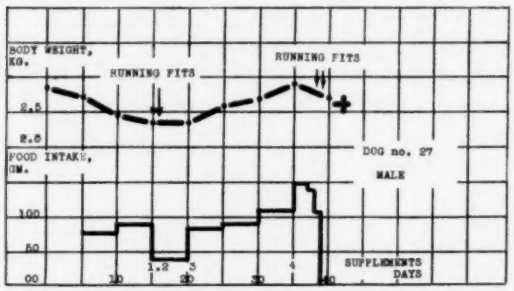

Fig. 1. Growth curve and food intake of dog 27 fed heated ration. Supplements: 1) 15 gm. of crude casein fed per day, 15th to 21st day inclusive; 2) 1 gm. of cod liver oil per day started 16th day; 3) daily allotment of casein at 10 per cent level placed on top of heated ration to insure intake; 4) casein decreased to 5 per cent of daily allotment.

Dog 27.—Dog 27 (fig. 1) was fed the heated ration as the sole article of diet. A severe attack of running fits occurred on the 15th day of the experiment. Fifteen gm. of crude casein was placed on top of the daily food allotment. The following day the dog suffered another attack of running fits but improved thereafter. Cod liver oil therapy was instituted.

On the 21st day 10 per cent of crude casein, based on the daily allotment, was placed on top of the diet. Although the pup was not growing at an exceptionally good rate, there were no indications of running fits two weeks later. The crude casein supplement was decreased to 5 per cent of the daily allotment. In three days the dog had a severe attack of running fits, stopped eating, and died suddenly.

Dog 25.—Dog 25 (fig. 2) was fed the heated ration as the sole article of diet. After 16 days on the ration it had a severe attack of running fits. It had lost weight steadily. Cod liver oil, thiamin chloride, and riboflavin were administered to guard against possible vitamin-deficiency complications. Five gm. of crude casein was placed on top of the ration.

The animal suffered a second attack of running fits the following day. It was induced to empty the stomach and appeared to be somewhat relieved but did not eat. On the 18th day of the study, in order to stimulate the appetite, dry canned dog food was given as the sole article of diet.

Four days later the food intake of the animal reached a significant level and a great improvement over the previous few days was noted. On the 23rd day we became aware that the heated ration was deficient in a number of essential factors.

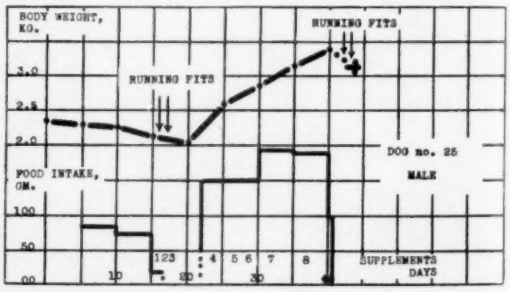

Fig. 2. Growth curve and food intake of dog 25 fed heated ration. Supplements: 1) 5 gm. of crude casein; cod liver oil, thiamin chloride and riboflavin; 2) the dog was induced to empty its stomach contents; 3) fed dry canned dog food as the sole article of diet but did not eat until approximately the 21st day; 4) dry canned dog food decreased to 50 per cent of the total ration; 5) supplement decreased to 33 per cent, then to 25, to 20 and, finally, to 10 per cent.

Therefore, we gradually decreased the amount of the canned supplement to a point where it would not prevent the onset of running fits but would improve the heated ration, as judged by food intake and growth. The level of the canned food was thus decreased to one part of canned food to one part of the heated ration.

At the end of three days on the above ration we observed that the animal was growing well. The dry canned food was decreased to 33 per cent of the total ration. On the 28th day the supplement was decreased to 25 per cent, and four days later

it was reduced to 20 per cent. On the 37th day the supplement was reduced to 10 per cent of the diet. At this level the canned food added 3 per cent of protein to the diet.

Five days later the animal suffered a severe attack of running fits. It was unable to retain food, had another attack the following day, and died.

Dog 28.—The animal (fig. 3) was started on the heated ration supplemented with 5 per cent of purified casein. On the tenth day the casein supplement was increased to 10 per cent, since the dog had not gained any weight. Three days following, the dog was rather ill but specific symptoms could not be observed. On the 16th day of the experiment cod liver oil, thiamin chloride, and riboflavin therapy were instituted to eliminate the complications of a vitamin deficiency. The 22nd day the casein supplement was placed on top of the ration to insure the intake of this component.

Since dog 25 was not protected against running fits when the heated ration was supplemented with 10 per cent of dry canned dog food, the diet of dog 28 also was supplemented with 10 per cent of dry canned dog food on the 43rd day. The daily portion of casein was again mixed into the heated ration.

At the termination of the second month of the experiment it was apparent that the dog was eating well and growing well. The casein component of the diet was therefore withdrawn at this time.

Eight days later the animal had an attack of running fits. After the one attack it appeared to be in fair condition. Since the dog was not eating well, no therapy was given.

On the 70th day of the experiment the dog had several severe attacks of running fits. One-half gm. of lysine dihydrochloride was administered orally at noon. The symptoms of running fits were not alleviated by evening. The following morning the dog was dead. It thus appeared that the therapy had been started too late.

Dog 26.—The animal (fig. 4) was fed the heated ration as the sole article of diet. On the eleventh day of the experiment it suffered a severe attack of running fits. The hemoglobin was 12.5 gm. per 100 cc. of blood. Five gm. of crude casein was placed on top of the baked food. At the end of the second week the dog had a severe attack of running fits. Ten gm. of crude casein was placed on top of the heated ration.

On the 15th day the animal had another severe attack of running fits. Ten gm. of crude casein was placed on top of the heated ration. The day following, thiamin chloride and riboflavin were administered to avoid vitamin-deficiency complications. The dog seemed to be weak. On the 17th day 10 gm. of crude casein was placed on top of the heated ration, and 15 gm. was given the next day. Another 15 gm. of the crude

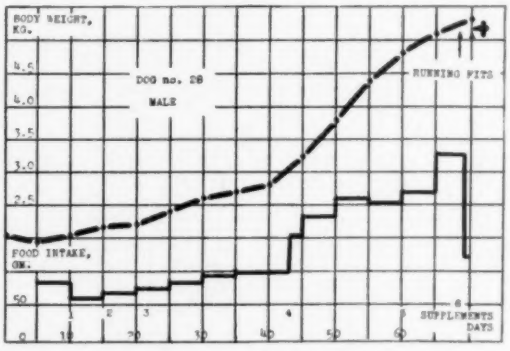

Fig. 3. Growth curve and food intake of dog 28 started on heated ration supplemented with 5 per cent of purified casein. Supplements: 1) purified casein supplement increased to 10 per cent; 2) cod liver oil, riboflavin, and thiamin chloride administered daily; 3) casein supplement placed on top of ration daily; 4) 10 per cent of dry canned dog food added to ration and both the canned food and casein mixed into ration; 5) casein supplement withdrawn; 6) 0.5 gm. of lysine. 2HCl administered.

casein supplement was given on the 19th day.

Twenty-four hours later 10 per cent of crude casein was added. The casein supplement was placed on top of the heated ration to insure its being eaten. In effect, the casein supplement was somewhat more than 10 per cent of the diet, since the entire amount of the heated ration allotted was rarely eaten.

At the end of the fifth week the dog was in good condition and the casein supplement was discontinued. One week later 10 per cent of dry canned food was added to the

ration. On the 54th day the dog was definitely not well. Irritation provoked an attack of running fits. Lysine dihydrochloride, 0.25 gm., was injected subcutaneously.

The next day the dog suffered another attack of this nervous affection. Lysine dihydrochloride, 0.45 gm., was fed and 0.15 gm. of the same amino acid was injected. There were two feedings and one injection at different times during the day.

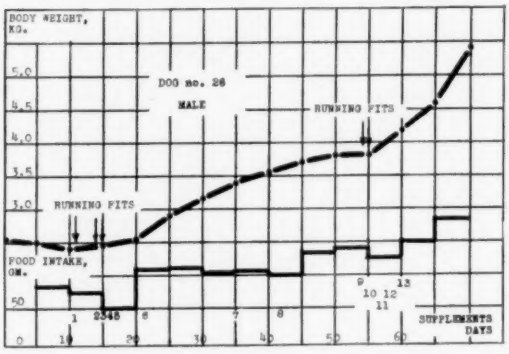

Fig. 4. Growth curve and food intake of dog 26 fed heated ration. Supplements: 1) fed 5 gm. of crude casein supplement; 2) 10 gm. of crude casein; 3) 20 gm. of purified casein; 4) fed thiamin chloride and riboflavin; 5) fed 15 gm. of crude casein 17th to 20th days, inclusive; 6) started daily supplement of 10 per cent casein placed on top of the daily allotment of heated ration; 7) casein supplement was discontinued; 8) 10 per cent of dry canned dog food mixed into ration daily; 9) injected 0.25 gm. of lysine dihydrochloride; 10) fed 0.45 gm. of lysine dihydrochloride and injected 0.15 gm. of lysine dihydrochloride; 11) injected 0.25 gm. of lysine dihydrochloride; 12) injected 0.25 gm. of lysine dihydrochloride; 13) added 10 per cent of purified casein to the diet daily.

No attacks of running fits were observed during the next 48 hours. The dog was weak but seemed to be in control of its faculties. Lysine dihydrochloride, 0.25 gm., was injected. On the 58th day of the experiment the dog was still weak but was otherwise normal. Lysine dihydrochloride, 0.25 gm., was injected.

The dog gained 400 gm. in the five-day period of lysine therapy. Since the supply of lysine was exhausted, a supplement of 10 per cent of purified casein was added to the diet on the 60th day. At the end of the tenth week the dog appeared to be normal and was gaining weight rapidly. By the 85th day the weight was 7,600 gm. and the animal was in good condition.

DISCUSSION

The results obtained with the dogs are summarized in table I. Dogs fed the heated ration as the sole article of diet developed the running fits condition in eight to 18 days. The evidence strongly indicates that running fits is a deficiency disease which may be corrected or prevented by the addition of an adequate amount of animal protein in the diet.

In these studies casein was used as the source of protein. The use of either crude or purified casein appeared to be equally effective in the prevention or cure of running fits. In several instances the response to casein appeared to be quite slow. Under these conditions the dogs had not ingested a sufficient amount of casein to be brought out of their moribund state.

The evidence obtained with dogs 25, 26 and 28 showed that using 10 per cent of dry canned dog food was insufficient to protect them against the disease. The dry canned dog food supplement did improve the heated ration to the extent that the food intake of the dogs increased and the rate of growth improved. This level of dog food added only 3 per cent of protein to the diet. Where casein was supplied at a low level, 5 per cent of the diet for dogs 22 and 27, the running fits condition appeared with fatal results.

The record obtained with dog 28 furnishes the best evidence for the conclusion that running fits is a deficiency disease. This dog received the heated ration supplemented with 5 per cent of purified casein for ten days and then supplemented with 10 per cent of purified casein for 50 days. During the last 17 days excellent growth resulted, due to the addition of a 10 per cent dry canned dog food supplement to the diet. At no time during the 60-day period did the dog suffer a severe attack of running fits. Eight days after the casein supplement in the diet was withdrawn, the dog developed this nervous affection.

The rapidity with which some of these dogs developed running fits when the casein

was withdrawn may indicate that there is an accompanying toxemia of some sort associated with the ingestion of the heated ration. Hogan, Guerrant and Ritchie[3] have noted an anemia in rats fed a deaminized casein. However, the hemoglobin reading on dog 26, at a time when the animal was suffering from a severe attack of running fits, was normal (12.5 gm. per 100 cc. of blood).

The results with casein have a number of important implications. In accord with the present concepts of protein chemistry, if casein corrects the deficiency, the active component must be one or more of the constituent essential amino acids. We attempted a preliminary study along these lines.

A number of investigators have already shown that when proteins are subjected to heat treatments, the nutritive value is impaired. Morgan[4] has reported that several cereals, wheat gluten and casein subjected to temperatures of 150° to 200° F. supported less vigorous growth and resulted in less favorable nitrogen retention than the materials which had not been heated. The digestibilities of the heated materials were found[5, 6] not to be impaired, although supplements of lysine and histidine were found to increase the nutritional value of heated casein but not of unheated casein. Mitchell,[7] Voegtlin and Thompson,[8] Block and coworkers[9] and Waisman and Elvehjem[10] reported beneficial effects when lysine was added to diets which contained proteins previously subjected to heat treatment. The third dibasic amino acid, arginine, recently shown to be essential on the basis of studies with chicks,[11, 12] has been reported to be ineffective.[10]

In accord with the findings of these investigators, lysine dihydrochloride was fed to dog 26 when it showed the running fits condition (fig. 4). That the treatment was beneficial may be seen from two related lines of evidence. The food intake of the dog did not drop, which was the case when the dog developed running fits at an earlier date. The growth of the dog during the period of lysine therapy was as good as when 10 per cent of purified casein was added to the diet. Thus, the efficiency of food utilization had not been impaired. The dog received the lysine dihydrochloride supplement for only a limited period of time, since our supply of the amino acid was exhausted. We propose to test both lysine and histidine as supplements to the heated ration to determine whether the feeding of these two amino acids will be sufficient to protect against running fits.

In this connection, the studies of Melnick and Cowgill[13] with high gliadin rations are of interest. These investigators observed running fits in dogs and stated, "Obviously,

TABLE I—Summary of results obtained with experimental dogs.

Dog	Diet	Time to Show Running Fits	Supplement Producing Cure
22	Heated ration only	8 days	100 gm. crude casein
	Heated ration + 5% crude casein	9 days	——
27 (figure 1)	Heated ration only	15 days	10% crude casein
	Heated ration + 5% crude casein	3 days	——
25 (figure 2)	Heated ration only	16 days	Dry canned dog food
	Heated ration + 10% dry canned dog food	5 days	——
28 (figure 3)	Heated ration + 10% purified casein	None in 60 days	——
	Heated ration + 10% dry canned dog food	8 days	——
26 (figure 4)	Heated ration only	11 days	10% crude casein
	Heated ration + 10% dry canned dog food	18 days	Lysine dihydrochloride; 10% purified casein

the phenomena were not due to the amino acid deficiency characteristic of the protein, for it was with the high gliadin diets alone that the reactions were observed." Our studies clearly show that running fits may be induced on diets with protein sources other than gliadin and that the deficiency may be alleviated or prevented by lysine-rich foodstuffs.

SUMMARY

A special lot of ration, processed by dry heat, consistently induced running fits when fed to dogs as the sole article of diet.

The condition of running fits was prevented by 10 per cent of purified casein in the diet for a 60-day period (dog 28). When the casein was withdrawn, the animal developed running fits in eight days.

A level of 10 per cent dry canned dog food was insufficient to prevent the onset of running fits. A level of dry canned dog food which supplied approximately 10 per cent of protein to the heated diet protected the dog against running fits.

Preliminary results indicate that the corrective action of protein is dependent largely upon the added supply of lysine.

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The bureau of census announced that the death rate for the whole United States last year was the lowest on record. There were 1,380,968 deaths, or 10.6 per thousand. The best previous record was that of 1933, when the rate was 10.7.

Formalin in Rickettsiosis

Intravenous injections of formalin are well tolerated in dogs in doses of 7 to 10 minims diluted in 10 cc. of normal salt solution. Two to three doses at intervals of 48 hours may be given, if found necessary, in grave septicemic infections. Pasquini (*Rec. de Méd. Vét.*, April, 1939) obtained excellent results from this treatment in rickettsiosis of dogs due to *Rickettsia canis*.

Spaying Bitches

Female dogs are deprived of their gonads for various reasons. Entire, they are a nuisance in the field and more or less troublesome at home on account of their biannual periods of estrum. Getting too fat is an objection raised against spaying but this is not as apt to occur when the operation is performed at an early age—before the ovaries have enlarged. If spaying is practiced before puberty or pregnancy, the bitch is no more apt to take on excess fat than any other pet. This statement is based upon over 8,000 observations. The operation should be performed at four to six months, or even earlier.

Estrual heat occurring after spaying is due to incomplete emasculation, and the treatment is removal of the remaining part of the ovary. The belief that estrum sometimes follows complete removal of the ovaries is not supportable. The contention (Dollar's Surgery, 1931) that estrum is not stopped by ovariectomy in some cases needs revision.

Removal of the horns down to and including a part of the body is preferable to removing the ovaries alone, because metritis and pyometra are prevented in the future. These post-operative reactions of the intact uterus are obviously due to lost tonus or the absence of certain ovarian secretions. *(H. C. Stephenson. A Discussion of Spaying Operations in the Dog and Cat. The Cornell Veterinarian, xxix, April, 1939, pp. 125-134.)*

Besides nitrogen and oxygen pure air contains carbon dioxide, helium, water, krypton, neon and xenon.

Urinary Calculi of Small Animals*

By H. C. STEPHENSON,† B.S., D.V.M.
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THE LOCATION of calculi within the urinary system is determined by their place of origin and their size. The pelvis of the kidney is the usual site in that organ. We have seen calcium phosphate calculi on autopsy in the tubules of the kidneys in one case, and calculi in the cortex of the kidney have been reported. These renal stones are rare in our locality, only two cases having been encountered in all of the autopsies at the small animal clinic of the college. Ureteral calculi also are rare. None were diagnosed and only a few cases showed sand within the ureters on autopsy.

On autopsy a case was observed in which a large quantity of sandy calculi had been removed from the bladder. Within twenty-four hours after the operation the dog died. A rim of sandy deposit was found around the opening of each ureter. The ureters and the pelvis of the kidney contained fine sand.

Although many of the cystic and urethral calculi form in the bladder, undoubtedly many form back up in the kidney and pass down to the bladder, where they gradually become enlarged unless soon washed out. The designations used to identify urinary calculi, such as ureteral calculi, renal calculi, cystic calculi and urethral calculi, are of use in indicating location but not origin. Whether the calculi found in the prostate are of urinary origin, we can not say, since we have seen only two prostatic calculi and, unfortunately, did not have them analyzed.

Calculi are also classified according to composition, *i.e.*, uric acid, calcium phosphate, cystin, calcium carbonate, and calcium oxalate. In addition to these, Milks of Cornell lists xanthin and the urostealth calculi, which are said to be derived from degenerated epithelial cells.

Calculi are rarely of pure composition. Frequently, they are mixtures, with the composition changing in different layers.

CAUSES

The causes of the formation of calculi are still somewhat of a mystery. Many theories, some at least partially proved, have been advanced. Consumption of food and water, especially rich in the salts that are most frequently found in calculi, has been cited as a cause. Most varieties of calculi contain calcium. Practically all diets contain calcium. If there were not some additional factor, all dogs and cats and, likewise, humans would be suffering from calculi.

A change in the chemical reaction of the urine has frequently been advanced as a cause. It is true, of course, that an alkaline urine in a dog favors the deposit of certain salts, such as the phosphates.

Concerning the formation of calculi, Glass states:

> They are generally started in their formation in the pelvis of the kidney, and, generally, from some foreign body, such as a blood clot, a piece of mucus, epithelium, etc., around which, if the urine is surfeited with salts that can be precipitated, the sediment in the urine forms and gradually the crystalline elements accumulate. This deposit is formed very quickly in cases of cystitis, where the urine is undergoing alkaline fermentation and produces a copious sediment in the urine.

Milks makes the following statement:

> The reaction of the urine may be important, but it is not the primary factor, since all alkaline urines do not form calculi and because phosphates are always deposited in alkaline urine. However, we know from clinical experience that an alkaline urine associated with certain infections, such as the micrococcus urease, which produces ammoniacal fermentations, is quite characteristic of inflammations of the bladder and the inflammatory condi-

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†Professor of materia medica and small animal diseases, Cornell University.

tions are frequently associated with calculus formation.

McCunn, the new editor of Hobday's "Surgical Diseases of the Dog and Cat," the fourth edition of which was published the past January, apparently is so impressed by the relationship of infection and calculi formation that he groups cystitis and stone under one heading.

We have been impressed by the fact that nearly all authors writing on urinary calculi stress the importance of a foreign substance being the nucleus of a calculi. Practically all dogs and cats at some time have these foreign bodies, such as clumps of bacteria, masses of epithelial cells, blood clots and renal casts, yet relatively few have calculi. Most of the calculi analyzed in our studies contained phosphates, which indicates an alkaline urine. Also, in most of our cases there was a cystitis. We were unable to determine if the calculi caused the cystitis or if the calculi were the result of the cystitis. On the other hand, we have encountered a number of cases of cystitis without calculi. If the cystitis had been allowed to continue without treatment, however, it is conceivable that calculi would eventually have been formed.

Within recent years, four other theories have been advanced to explain the formation of urinary calculi. They are:

1) The rôle of focal infection. Rosenow and Meisner infected devitalized teeth in dogs with streptococci of human origin from patients with urinary stone. In the majority of cases, they found that urinary stones formed in the dogs.

2) Vitamin A deficiency. McGarrison found that unbalanced diets, containing whole wheat and oatmeal, tend to produce stone in white rats. He found that this tendency could be overcome by sufficient vitamin A. We treated two cases of calculi in young pups that have not had a recurrence for over two years, in which our after treatment consisted of a substantial supply of vitamin A.

3) Calcium-phosphorus imbalance. McGarrison also determined that a low blood phosphorus and a high blood-calcium level favor the formation of stone. Barney and Mintz reported a number of human cases of stone associated with hyperparathyroidism. However, they did not credit the formation of the stones entirely to the low blood-phosphorus and the high blood-calcium levels. They theorized that infection, vitamin deficiency and changes in the urinary colloids are important factors.

4) Urinary colloid imbalance. The urine may be considered as a colloidal solution which holds in solution much more material than otherwise could be contained in an aqueous solution. Lichtwitz and Keyser feel that changes in the colloid content of the urine largely determine the precipitation of the crystalloids. Some error in metabolism and infection is believed to affect the colloidal balance.

The above discussion would indicate that very little is known about the formation of stone, but there seems to be some agreement that infection, unbalanced diet, and vitamin A deficiency are important factors.

SYMPTOMS

The symptoms presented by a case of urinary calculi either in the dog or cat may range all the way from those of a mild cystitis to a complete stoppage of the urine. Frequently, a calculi will attain a great size before it is observed by the owner. The x-ray (fig. 1) of three large calculi in the bladder of an aged female Poodle serves to illustrate this point. The owner claims to have noticed straining only one week before he brought the animal to the clinic. The stones were smooth and we found during the operation that the bladder wall was not greatly thickened.

Frequent desire to urinate, especially in the house dog, is a common history. Also, the squatting of males to urinate may be the only history. Most owners are unqualified to associate cause and effect in animals and, therefore, any history, from constipation to worms, does not surprise us. Abdominal pain caused by distention of the bladder has led more than one owner to decide his dog has worms. The most indicative history of all, of course, is the one that tells us that the dog or cat urinates

frequently and passes a little blood in the urine, especially in the last few drops.

DIAGNOSIS

All of the above histories are not exactly indicative of calculi but they are indicative of what we should examine. Diagnosis depends upon examination. Chemical examination of what voided urine we may obtain will ordinarily be of little help. It will probably indicate a cystitis but will not tell us definitely that there is or is not a stone. We must examine the animal itself if we are to be sure of our diagnosis.

If the abdomen is distended, we should first try ballotment, to determine if the bladder is full. This is done by holding one hand flat against one side of the abdomen and tapping the opposite side with the fingers of the other hand. If there is free fluid within the abdomen, a wave passing through the abdomen can be felt by the flat hand. The force of the tapping fingers will be delayed and felt by the flat hand a fraction of a second later. If the bladder is full and intact, this wave will not be felt. If the bladder has ruptured, the wave will be felt. Of course, if the free fluid is ascital, or peritonitis exudate, a wave will be felt. Aspirating some of the free fluid will allow a determination of its nature. Also, if the bladder has ruptured, the general condition of the patient will be that of collapse.

As long as the bladder is distended with urine, it will be virtually impossible to determine the presence of a stone. In many cases the bladder will be nearly empty. If such is the case, palpation of the bladder through the abdominal wall should be attempted. If the case is of long duration, a thick-walled bladder will probably be felt. In some cases it may be necessary to pass the index finger of one hand into the rectum. With the other hand pressing upward on the abdomen in front of the publis, it may be possible to feel the bladder. Frequently, the presence of stones can be determined in this way.

In the few cases we have encountered in cats, the bladder could easily be palpated through the abdomen. If there is complete stoppage of urine in the cat, the bladder may be round and very firmly contracted so that its walls can not be rolled between the fingers. It is usually by this rolling of the bladder between the fingers that we can determine the presence of the single, medium-sized stone or the many small stones or sand, both in the dog and the cat.

Figure 2 shows a number of calculi in the bladder of a castrated male cat. This animal was brought to our clinic last January. There was complete stoppage of the urine and the bladder was round, very hard, and could not be compressed. We were unable to pass a catheter. After the urine was withdrawn, by puncture of the bladder through the floor of the abdomen, the stones could be felt within the bladder, like marbles or BB shot within a bag. When the bladder was empty, we were able to pass a catheter.

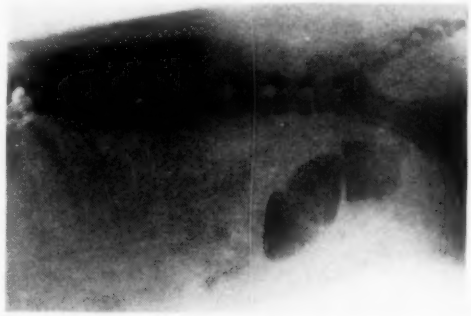

Fig. 1. Aged female poodle. Stones were smooth. Owner noticed straining only one week before bringing animal to the clinic.

The passage of a metal catheter in the bitch can be of great assistance in the diagnosis of either urethral or cystic calculi. We have not been successful in passing a catheter in the female cat, possible because we have encountered very few cases requiring this. We have not observed complete stoppage of the urine in the female cat, and the type of stones found were of the sandy variety. They did not cause a plugging of the urethra of the female cat.

In the bitch, we use a 12-inch human male catheter, size 8 or 10. We straighten this a little. The usual short, metal, human female catheter sold to veterinarians has proved very unhandy in our experience. We dilate the vagina with a Sim's rectal specu-

lum in large dogs or a human urethral speculum in small dogs or spayed bitches and we use a light to find the urinary meatus about 1 inch or more anterior to the clitoris, on the floor of the vagina. Although at times we have passed a catheter in the bitch without using a speculum and light, this procedure has resulted in failure in 90 per cent of the cases.

Diagnosis of either urethral or cystic calculi in the bitch is confirmed by contact of the metal catheter with the stone or stones. The lower half of figure 3 shows a metal catheter that has been passed into the bladder of a small, smooth-haired Fox Terrier. She was brought to the clinic the last of January of this year with a history of pus in the urine and frequent urination.

Fig. 2. X-ray of fine stones in bladder of castrated cat. The stones show as a cloud.

Methenamine, 5 grains, was dispensed, to be used twice daily. The owner brought her back about March 15 with a further history of frequent urination, straining, and the occasional passage of blood in the urine. A metal catheter was passed and a diagnosis of calculi was made when the catheter contacted the stones.

The lower half of figure 3 shows the catheter inserted. We were able to feel the stones but they are not clear in the x-ray picture. We attached a length of rubber tubing to the catheter and inflated the bladder and made the exposure shown in the upper half of the illustration. If the urine is withdrawn from the bladder and some air blown in, the stones will throw a much more distinct shadow on the film. This film demonstrates that at least in the bitch an x-ray is not required to make a diagnosis. The stones were felt by means of the catheter. It has not been our practice to make the diagnosis of calculi by means of the x-ray. We had heard that stones do not show by x-ray and, therefore, became curious and took some pictures.

After removal of the stone by cystotomy, the cystitis was treated with methenamine. Twelve hundred units of vitamin A were administered daily for over a month. There has been no recurrence at the time of writing (June).

The diagnosis of cystic or vesical calculi in the male dog can not easily be made by means of a catheter. We do not know of a metal catheter that can be passed through the os penis of the male dog and around the ischial arch into the bladder. We have tunked against a large calculus in the bladder of a male dog with a French web catheter but do not have much confidence in this method. If we think there might be a calculus but can not confirm it by palpation as previously described, we anesthetize the animal. The chances of palpating calculi in the bladder are much greater when the abdomen is completely relaxed. X-ray can be utilized.

Before considering bladder and urethral stones in the male cat, we will describe the case illustrated in these last films. The patient was a 3-year-old black Cocker, male. The owner had noticed frequent urination and straining for three days. We took the dog to a post in our back yard and, when he attempted to urinate, we held out a dipper, receiving 4 ounces of very dark urine. In the bottom of the dipper we discovered twelve sandy particles.

A catheter could be passed only as far as the posterior end of the os penis, where it was stopped by a calculus. This calculus was removed through an incision into the urethra. While the dog was under anesthesia, the bladder was palpated and the stone shown in figure 4 was discovered. Figure 5 shows the cystin calculi removed from the bladder. When the urethral incision healed, the bladder stone was removed by cystotomy. The first urine sample taken showed it to be composed mainly of cystin. After the incision healed, the owner was advised to reduce the meat ration a

little. Otherwise, we could offer no advice as to how to prevent a recurrence. This case came to us the past January and, as far as we know, there has been no recurrence.

We have never encountered any large stones in the bladder of the male cat. The deposits always were of the nature of fine sand or the size of BB shot. In most of the cases there was complete stoppage of the urine. All of our cases of male cats having calculi were castrated. We do not recall an entire mature male cat or a male that was castrated after maturity having been brought to us suffering from calculi. Perhaps this proves only that calculi in cats are relatively infrequent in our locality. It also suggests that if the penis is fully developed, the urethra is larger and, hence, sandy calculi are more easily passed.

To determine if there are calculi in the penis of a cat, we pass either a straight metal catheter of the type designed by Dr. Frink of Los Angeles, or a whalebone bougie. These whalebone bougies can be obtained in very small sizes. The olive points are smooth and, therefore, there is little danger in using them if the penis can be protruded in a straight line with the floor of the pelvis. Manipulation of the bougie may facilitate the release of the fine sand or force some of it back into the bladder. In using the metal catheter we frequently attach it to a syringe and squeeze a little boric acid solution into the urethra. This may help to straighten out the urethra and wash out the deposits.

On occasions we have drained the bladder by puncturing the abdominal wall. After doing this we were able to pass the catheter. Others have had the same experience. This is difficult to explain but it appears that the full bladder creates some reaction in the urethra that prevents its easy dilation, or else the neck becomes slightly bent so that no urine is passed.

The diagnosis of urethral calculi in the male dog is relatively easy. All that is needed is a metal probe. After protrusion of the penis, insert the probe in the urinary opening and pass backward. The stone will usually be felt at the posterior end of the os penis. It will feel like tapping against a rough stone or like rubbing a probe over a rough stone. If the peculiar grating is not felt, it will be well to pass a catheter as far as the bladder. We prefer the French web with the olive tip, as a stylet is not necessary.

Many times, when there is complete stoppage of urine, a catheter can be passed by the stone so that distention of the bladder can be relieved. In some of the large breeds of dogs, the ordinary human-size catheters may be too short. If one of the Whipple type dog catheters is not available, a human urethral catheter can usually be obtained from the drug store. This will be long enough for the largest dog.

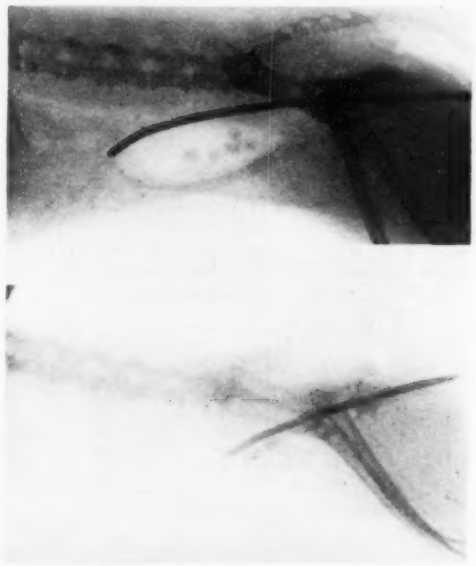

Fig. 3. This x-ray study shows metal catheter in bladder of a small smooth-haired Fox Terrier. The diagnosis was made by contact of catheter with stones. Upper half shows stones clearly, since the urine had been drained out and the bladder was distended with air.

For a long time attempts have been made to relieve humans who have stone and yet refuse the dangers of an operation. Similar attempts have been made on dogs and cats. There have been many disappointments. The latest prescriptions that have come to our attention appeared in an article by Dr. C. P. Zepp of New York City in the New York State Veterinary Society News Letter for April, 1939. We have not used

them, but have been assured by Dr. Zepp that he has had success with them. The prescription to be used in case of acid urine is as follows:

Potassii Citrates	℥j
Sodii Bicarbonates	℥jv
Syr. Aurantii	℥jv
Aquae Destillatae q. s.	℥viij
Mft.	
Sig. One dram tid.	

Fig. 4. Cystin stone in male Cocker. The urine had been withdrawn and the bladder was partly distended with air. A previous x-ray picture taken without distending the bladder with air failed to reveal a stone.

In the case of alkaline urine a different prescription is recommended:

Ammonii benzoatis	℥ij
Ammonii chloride	℥iij
Sacrosi	℥jv
Elixir pepsini lactati q. s.	℥viij
Mft. Sympus	
Sig. One dram tid with water.	

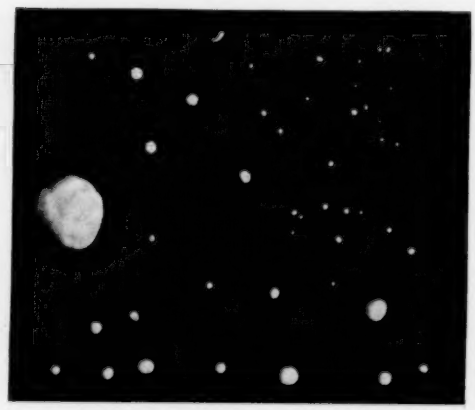

Fig. 5. Cystin calculi removed from bladder of male Cocker.

CYSTITIS

Cystitis is present in some degree in all cases of calculi. Some observers believe that it is the actual forerunner of the formation of calculi. Whatever may be its rôle, it can not be ignored. Washing the bladder with warm 1 per cent boric acid solution can be particularly useful. It is soothing to the mucous membranes, as well as somewhat bacteriostatic. Flushing out the bladder also aids in removing fine, sandy deposits. If the bladder wall is greatly thickened, rather small amounts of boric acid should be used. The use of boric acid orally is dangerous because of the gastroenteritis produced. Other drugs that can be used for flushing the bladder are the organic silver compounds, such as 1 per cent protargol and 20 per cent silvol.

The past winter we treated a case of sandy calculi in a castrated male cat by daily flushing out the bladder with about 10 cc. of 1 per cent boric acid solution. For ten days this cat could not urinate of his own accord. Each day we passed a metal catheter, drained the bladder, and then irrigated. On the eleventh day he urinated a little. From then on we could squeeze out a little urine. We continued the flushing of the bladder every third day for three more weeks, after which he made a recovery. The owner reported in the middle of May that the animal was apparently in good condition. Internally, we gave 5 grains of methenamine twice a day.

Urinary antiseptics that can be used *per os* are methenamine (urotropin, cystoform, ammoform, hexamethylene-tetramine), if the urine is acid; caprokal (hexaresorcinol in oil) for both acid and alkaline urines; some of the azo dyes, such as pyridium, which is effective against *Bacillus coli*, and serenium, which is more effective against the cocci. Caprokal appears to be more effective against the cocci than against *B. coli*. Sulfanilamide also may be used as a urinary antiseptic, if there are streptococci present.

Treatment of cystitis by the internal administration of urinary antiseptics offers an ideal method of attack. Naturally, it has its limitations, due to the character of the urine and to the fact that the drug must be very potent to be still in such concentration as to produce an antiseptic effect or

bacteriostasis. Flushing of the bladder with proper solutions and with reasonable care should be considered as a proper adjunct to internal administration.

Figure 6 illustrates the thickening of the bladder in cystitis accompanying calculi. The bladder does not distend normally.

RECURRENCE

The control of the recurrence of calculi is still a mystery. In most of our cases in which there was a recurrence, we also were dealing with a chronic cystitis. We feel that our inability to bring about normal bladder conditions has been an important factor. In a few cases of calculi in young puppies, recurrence was apparently prevented by adding vitamin A to the diet. Of course, we are not certain that there would have been a recurrence. Some observers believe that controlling the acidity or alkalinity, depending upon the type of stone removed, is of benefit. It has been recommended that when the stone is of the cystin or uric acid type, a low meat diet will prove effective, and that when the stone contains calcium salts, an increased meat ration will be of benefit.

In many cases, recurrence of calculi can not be controlled. The animal seems to have reached perfection in its ability to produce calculi and all our efforts in prevention are of no avail.

PROGNOSIS

The prognosis is grave for untreated cases of calculi. Where there is complete or partial stoppage of urine, prognosis is grave unless relief is secured promptly. Surgical interference is indicated immediately; at least, some measure, such as catheterizing or puncturing the bladder, should be performed. Once the danger of collapse is over, further surgical intervention can be postponed for a few hours, until the examination has been completed and a further procedure determined.

In the case of cystic calculi, there is little danger from an operation unless the cystitis is severe. When the walls of the bladder are thickened, it is more difficult to sew up the bladder incision properly, and the danger of peritonitis is greater.

In the case of urethral calculi, there is ordinarily little danger from the operation, although we did lose two cases from infection. Care, in these cases, should be directed toward the prevention of stricture by passing a catheter or sound daily.

Fig. 6. This x-ray illustrates the pronounced thickening of the bladder that may occur in cystitis accompanying calculi.

CONCLUSIONS

The cause of urinary calculi is complex, as evidenced by the number of theories advanced in explanation. The size of the calculi determines the chances of urine stoppage. Renal and urethral calculi are diagnosed very infrequently, although the source of calculi found elsewhere may be in the kidney. The principal symptom is the frequent attempts at urination. Blood in the urine is a symptom of the presence of calculi. No symptom in itself is conclusive; it simply indicates where to look.

Diagnosis depends upon examination. Examination is not complete without catheterization. Catheterization of the castrated male cat may be difficult. The cystitis accompanying calculi requires attention. We have yet to learn, in the majority of cases, how to prevent a recurrence, although we realize the apparent importance of vitamin A. Until some certain method of prevention of recurrence is found, stone must be considered a serious condition.

I have never obtained any benefit from the use of sulfanilamide in the cerebrospinal complication of canine distemper.—*T. B. Crowe, D.V.M.*

Vitamin A Deficiency in the Dog. 1. Experimental Production of the Vitamin A Deficient Condition*

By WALTER C. RUSSELL† and M. L. MORRIS,‡ B.S., D.V.M.
New Brunswick, N. J.

SINCE the production of a vitamin A deficient condition in the dog by Steenbock, Nelson, and Hart in 1921,[1] a number of investigators have produced the deficiency in this species. The present report describes the effect of the lack of vitamin A in dogs and the results of certain blood studies that were made during the development and cure of the deficiency.

EXPERIMENTAL PROCEDURE

The vitamin A deficient ration was a modification of that used for the production of vitamin D deficiency, which will be described in a later paper of this series. The ration consisted of (per cent) rolled oats, 54.2; casein (extracted with 95 per cent alcohol for 15 hours to remove vitamin A), 8.4; meat scrap (55 per cent protein), 8.4; cane sugar, 9.3; dried yeast, 8.4; Crisco, 5.4; calcium carbonate, 3.4; calcium phosphate (tertiary), 1.7; and sodium chloride, 0.8.

At first, the ration was prepared by adding the above constituents, except dried yeast, to boiling water, and the mixture was stirred and cooked until a thick pasty consistency was obtained. The dried yeast was stirred in as the mixture cooled. When cold, the ration had the consistency of cooked rolled oats that had set to a stiff mass. The ration prepared in this manner was too pasty and was eaten by the dogs with difficulty. After several experimental trials it was found that a dry ration, when slightly moistened, was readily consumed. Consequently, the rolled oats were cooked as if for human use, dried in an oven and ground. This product was mixed thoroughly with the dry constituents of the ration and the melted Crisco added. When slightly moistened, this form of the ration was readily accepted. In the early stages of the experiment 4 to 8 ounces of raw meat was fed weekly to improve further the palatability of the dry ration.

As a supply of vitamin D, 330 mg. of irradiated dry yeast was administered by capsule three times a week. This quantity provided 500 U.S.P. units of vitamin D per day and served to protect the dogs against rickets. It was, however, probably somewhat in excess of the actual requirement. Although dried yeast was included in the ration as a source of the vitamin B complex, it was deemed advisable to administer thiamin chloride (vitamin B_1), as will be indicated later. Likewise, as will be discussed later, when an anemia was detected, iron ammonium citrate containing one part per thousand of copper sulfate was administered by capsule.

Dogs 98 and 99 were litter mates from a litter of mongrel Shepherd puppies, weaned at about 6 weeks of age. The diet and physical condition of the bitch were not known and the general appearance of the puppies was not good. In order to put the animals into good condition before placing them on the deficient ration and yet not permit them to acquire too great a store of vitamin A, they were fed a cereal food and a puppy-weaning preparation for one week.

Microscopic examinations of the feces were made and vermifuge was administered as indicated. Homologous anti-canine distemper serum was administered at frequent intervals. The dogs were housed in the noncontagious, surgical ward of a hospital.

RESULTS AND DISCUSSION

Dog 98.—As indicated in figure 1, this

*This is the first of a series of four papers which will describe the experimental production of vitamin deficiencies in the dogs which were exhibited at the 75th annual meeting of the A.V.M.A., New York, N. Y., July 5-9, 1938.

Journal series paper of the New Jersey Agricultural Experiment Station, department of agricultural biochemistry.

†New Jersey Agricultural Experiment Station and Rutgers University.

‡Raritan Hospital for Animals.

animal gained during the first 22 days on the deficient ration. The marked decrease in growth rate which followed seemed to occur too early as a manifestation of vitamin A deficiency, and vitamin B_1 deficiency was suspected. Upon the administration of 1 mg. of vitamin B_1 (thiamin chloride) there was an immediate response. Daily feedings of 1 mg. of this vitamin were continued for nine days, after which it was supplied at intervals of several days.

Following the first feeding of vitamin B_1 the animal grew rapidly for six days, after which the body weight tended to become constant, despite further supplementation with this factor. The 1 mg. per day dose of vitamin B_1 (300 International units) was greatly in excess of the actual requirement, but this large amount was fed as an emergency measure in order to prevent the loss of the dog. The feeding of iron and copper was begun five days before the administration of vitamin B_1 and, as discussed in the section on blood studies, may have contributed to the growth response. Nine days of practically constant weight and the condition of the animal indicated that a vitamin A deficient condition had developed. This condition was reached 45 days after the feeding of the vitamin A deficient ration was begun.

Since it was desirable for exhibition purposes to show one dog in which the vitamin A deficient condition had been developed and in which a cure of the deficiency was in progress, dog 98 was fed, at first, a quantity of carotene (provitamin A) approximately seven times the minimum requirement of 90 U.S.P. units of carotene per pound of body weight per day given by Frohring.[2] The carotene administration began with 3,300 U.S.P. units per day (630 units per pound). After several days of feeding at this level there was no marked growth response. Therefore, larger doses were administered, in some instances as large as 18,000 U.S.P. units per day in the form of a fish liver oil preparation.

Despite the large doses of vitamin A, both in the form of carotene and of preformed vitamin A, dog 98 did not show as marked a growth response as did dog 99, which received a much smaller daily dose. Why this animal did not show a marked growth response is not known. Its supply of vitamin A may have been too severely depleted and, in consequence, its ability to utilize vitamin A efficiently may have been impaired. The blood picture, discussed in the section on blood studies, indicated an infection which may have retarded growth. Fourteen days after the feeding of the vitamin supplementation was begun, dog 98 resumed growth and it continued to grow to the termination of the experiment at essentially the rate shown during the first

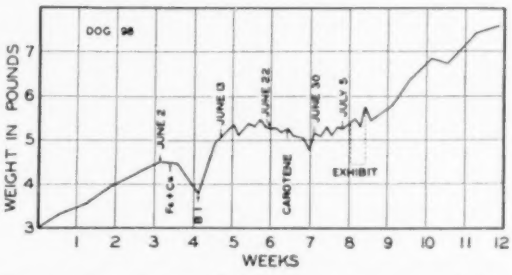

Fig. 1. Growth rate of dog 98 on experimental diet.

three weeks on the vitamin A deficient ration.

Dog 99.—This animal presents essentially the same history as dog 98. The vitamin A deficient condition was pronounced in 47 days (fig. 2). The condition developed before the time that the animal was to be placed on exhibition and, consequently, it was necessary to administer carotene or vitamin A in order to prevent the loss of this animal.

A photograph (fig. 3) was taken on July 2, four days after the beginning of the carotene feeding but before the dog had begun to respond to the supplement. It shows the deficient condition of the animal. Frohring[2] has reported the minimum daily curative dose which would effect a definite increase in body weight to be 90 U.S.P. units per pound. Since it was our desire only to maintain body weight, it was estimated that 60 U.S.P. units per pound should be fed. Four daily doses of carotene at this feeding level were administered. The dog did not respond immediately to the feeding of carotene and, in order to save

the animal for exhibition purposes, it was necessary to resort to artificial heat, injection of glucose and saline, and the feeding of a puppy-weaning food which contained some carotene.

Six days after the first dose of carotene, growth was resumed and continued, although there was no further supplementation, for 36 days, after which the experiment was terminated. During this period the animal gained 3 pounds 3 ounces, as contrasted with 2 pounds 5 ounces for dog 98 for essentially the same period of time. Since the latter animal received many times

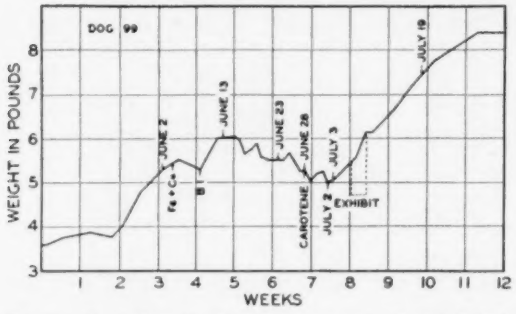

Fig. 2. Growth rate of dog 99 on experimental diet.

the amount of the factor fed No. 99, yet did not grow as well, it is possible that both animals grew at what might be considered their normal rate. However, a litter mate, dog 94, on the same type of ration, which received adequate vitamin A but lacked vitamin D, weighed 18 pounds 4 ounces when dog 99 weighed 7 pounds 8 ounces, 21 days after receiving the first carotene supplement.

The difference in weights of the dogs at the start of the experimental periods was 1 pound. Figure 4 shows dog 99 (the smaller of the two dogs) 21 days after the first feeding of carotene, and its appearance in this photograph should be compared with that in figure 3. Also, in figure 4 is shown a photograph of the litter mate, No. 94, on the vitamin D deficient ration.

The possibility of the retardation of growth rate due to infection is discussed in the section on blood studies.

BLOOD STUDIES

It was noted, after the animals had been on the deficient diet for 22 days, that their general condition was not entirely satisfactory. When blood examinations were made, certain deviations from the normal were noted in the blood picture (tables I and II), particularly with regard to the lowered hemoglobin and the actual reduction in the number of red blood cells. Consequently, the feeding of 300 mg. of iron ammonium citrate, with 1 part per 1,000 of copper sulfate, was initiated as a supplement to the vitamin A deficient diet.

After eight days of feeding the iron and copper supplement, the red blood-cell count and the hemoglobin value showed a definite increase. Eighteen days after the beginning of iron and copper feeding, a further increase in hemoglobin and red blood cells had occurred in both dogs. The hemoglobin content and red cell count then remained practically constant and were essentially the same as those observed for average normal puppies of this age.[3] Since vitamin B_1 was administered at practically the same time as the iron and copper, it is not possible to state whether the growth response was due solely to vitamin B_1 and the changes in the blood picture solely to the iron and copper, or whether the factors were effective in conjunction with each other.

It should be noted in the case of dog 98 that from June 22 to June 30 there was a slight increase in the hemoglobin and a marked increase in the number of red blood cells. This increase occurred at the plateau period, or at the time when the vitamin A deficiency was manifest. In the case of dog 99, during the period from June 23 to July 3, or the time that the animal was actually in a state of vitamin A deficiency, there was an increase in both hemoglobin and red blood cells and a concomitant rise in the color index. Changes in the red blood-cell count or the hemoglobin content of a pathological character were therefore not observed in either of these animals at the time of the deficiency.

Crimm and Short[4] reported low hemoglobin contents and red blood-cell counts in their vitamin A deficient dogs and they attributed the low values to a vitamin A deficiency. In view of the findings reported above, the question is raised as to whether

the low values reported by these investigators were due to the vitamin A deficiency or to a deficiency of iron and copper in the ration that they used.

These authors have also directed attention to certain characteristic changes which

Fig. 3. Dog 99, photograph taken July 2, 1938, when in a vitamin A deficient condition. Note general debility, unhealed abscess on shoulder and purulent condition of the right eye.

occurred in the differential leukocyte counts of dogs in a state of vitamin A deficiency. They have reported a characteristic "lag" in the Arneth index and a definite "left shift" in the Schilling index. They state, "This indicates a degenerative change in the granulocytes in A-avitaminosis." If the total white and differential blood pictures of dogs 98 and 99 are compared with the average normal for puppies of the same age, reported by Morris, Allison and Green,[3] it is apparent that on June 2 there was an increase in neutrophiles and also some decrease in lymphocytes. Although the white cell pictures of puppies is always somewhat variable, it is interesting to note that a definite "shift to the left" in the Schilling index occurred, characterized by a marked increase in the numbers of mature neutrophils and an actual reduction in the numbers of lymphocytes. In the case of dog 98, however, following the administration of vitamin A there was a decrease in neutrophils and a marked increase in the lymphocytes. Dog 99 did not show the same shift after June 23. On July 3, when this dog was in an advanced state of avitaminosis, the count showed 92 per cent neutrophils and 5 per cent lymphocytes.

The analysis and interpretation of these data are difficult, since dogs suffering with virus or mixed bacterial infections, seen in the clinic, show similar blood pictures, although their diets are known to contain adequate quantities of vitamin A.

In examining the report of Crimm and Short it was noted that one dog showed a chronic respiratory involvement for several weeks. The question arises, then, as to whether the striking changes observed in the white cell picture are significant of a vitamin A deficiency, or whether they are produced by a superimposed virus or bacterial infection.

GENERAL DISCUSSION

It is worthy of note that the loss in weight early in the depletion period might easily have been considered to be a condition of A-avitaminosis from the growth standpoint. Just prior to the time that the loss of weight was observed, the hemoglobin and the red blood-cell count were found to

Fig. 4. Dog 99 (right), photograph taken July 19, 1938, after growth had resumed on same diet with a vitamin A supplement added. Contrast the general appearance and eye condition with that in figure 3. Dog 94 (left) is a litter mate which received a diet adequate in vitamin A but deficient in vitamin D.

be subnormal. Following the administration of iron and copper and of vitamin B_1, there was a resumption of growth. Whether this was due to the vitamin or mineral supplement, or to both factors, is not known.

TABLE I—Blood findings, dog 98.

	6-2-38	6-13-38	6-22-38	6-30-38	7-5-38
Hemoglobin	43% 7.3 gm.	52.4% 8.9 gm.	64.7% 11 gm.	68.2% 11.6 gm.	64.1% 10.9 gm.
R. B. C.	3,900,000	4,360,000	5,030,000	6,260,000	6,030,000
Color index	0.55	0.60	0.64	0.55	0.52
W. B. C.	10,700	10,100	8,400	9,000	12,800
Achromia	Marked	Marked			
Juvenils			1% 84		
Stabs	2% 210	5% 505	5% 420	1% 90	
Segments	69% 7,383	76% 7,676	82% 6,888	70% 6,300	62% 7,936
Lymphocytes	23% 2,461	12% 1,212	7% 588	20% 1,800	19% 2,432
Eosinophils	3% 100	6% 606	2% 168	5% 360	17% 2,176
Basophils				2% 180	
Monocytes	2% 214	1% 101	3% 252	2% 90	2% 256
Normoblasts	1% 100				
Body weight	4 lbs. 8 oz.	5 lbs. 11 oz.	5l bs. 4 oz.	5 lbs. 3 oz.	5 lbs. 4 oz.

TABLE II—Blood findings, dog 99.

	6-2-38	6-13-38	6-23-38	6-28-38	7-3-38
Hemoglobin	34.7% 5.9 gm.	47.5% 8.1 gm.	61% 10.4 gm.	53.8% 9.1 gm.	66.4% 11.3 gm.
R. B. C.	2,960,000	4,410,000	5,210,000	5,650,000	5,320,000
Color index	0.59	0.53	0.59	0.48	0.63
W. B. C.	14,900	8,000	9,800	13,500	18,800
Achromia	Marked	Marked	Marked		
Stabs	3% 447	1% 80	3% 294	1% 135	1% 188
Segments	75% 11,175	74% 5,920	81% 7,938	72% 9,720	91% 17,108
Lymphocytes	15% 2,235	17% 1,360	11% 1,078	19% 2,565	5% 940
Eosinophils	6% 894	7% 560	4% 392	4% 540	3% 564
Monocytes	1% 149	1% 80	1% 98	4% 540	
Body weight	5 lbs. 5 oz.	6 lbs. 0 oz.	5 lbs. 8 oz.	5 lbs. 4 oz.	5 lbs. 1 oz.

When carotene and vitamin A supplements were given to dog 98, the presence of infection probably delayed the resumption of growth.

The experience in producing and curing vitamin A deficiency in dogs serves to emphasize the necessity of supplying a diet adequate in all required factors, except the one under investigation, in order that a clean-cut, specific deficiency can be produced. It is also important that frequent clinical examinations of the animals be made.

SUMMARY

The vitamin A deficient condition was produced in young dogs in 45 to 47 days.

A low hemoglobin content and a low red blood-cell count were not due to a lack of vitamin A.

The failure of the dogs depleted of their vitamin A stores to grow immediately after the administration of the vitamin A preparation may have been due to an infection.

It was not possible to determine whether the total white and differential blood picture was due to infection or the vitamin A deficiency.

The results emphasize the necessity of supplying a diet adequate in all required factors, except the one under investigation, and of frequent clinical examinations of the animals during the experiments.

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The Small Animal Branch of Veterinary Medicine*

THE LAST TIME I had the good fortune to attend a meeting of your association was 38 years ago, when on returning from the annual meeting of the national association in Atlantic City, the delegation from the Middlewest dropped off at Ithaca to participate in your program of 1901. So, my coming to a New York State meeting after all of these years of an itinerant career among the associations is a feature event of my life, for whatever we may claim for ourselves in the Central West, we are not unaware of the fact that it was the Law's, the Bell's, the Liautard's, the Moore's and other dominant figures of the Eastern Seaboard who set the standards for our conduct during the days when our profession was suffering from the growing pains of a sickly adolescence.

Objectives of Veterinary Pioneers Cited

It has been a great advantage to have followed the morals and ambitions inspired by those men among the scattered personnel who were to develop the man power of the important profession we here represent. They were proud professionals, those early New Yorkers, scholarly, capable, farseeing men who honored the Oath of Hippocrates without losing sight of the economic and public health sides of their tremendous undertaking. To the veterinary service they were launching, the United States was an unchartered sea. It required men of courage, brain and moral stamina to launch a profession from the material in sight. Yet, those forefathers, with the aid of their sparse contemporaries on the other side of the mountains, planted the seed that was to grow into the vast conservation service now commanded by the veterinary profession.

Theirs was a life of contemplation, of foresight. They were the architects of a service now recognized as indispensable to the progress of our country, of the structure we of the succeeding generation were entrusted to erect and in which you of this generation now live. I can not too often repeat that theirs was a difficult task—a task, I fear, the young men of this day are apt to overlook. In the language of sport, these pioneers started from scratch, *to wit:*

a) They founded an educational system;
b) they organized the associations;
c) they enacted laws and regulations; and
d) they established a literature.

To understand the house in which we now live, that is to say, the veterinary profession of the present time, one must know the conditions of 1875 and 1939 and all that transpired between these dates. While this is not a sketch on history, everyone of us should keep uppermost in mind that what we now glorify as the veterinary service of our country was started from absolutely nothing by these pioneers of New York. They set down standards of conduct which, although difficult to maintain in the rapidly growing profession, are the standards which have provided an honored and indispensable occupation, standards which we can not afford to set aside today nor cease to venerate and protect against dubious ethics, for therein lies our future rating among other branches of science and our usefulness to our country. All hope of a glorious future for the profession diminishes with every violation of the code of ethics set down by those forefathers to govern the behavior of professional men.

Influence of the Educational System

As our country tripled in wealth and population, the schools these pioneers founded furnished the first personnel for the military and civil service, manned the colleges, supplied men for research and sanitary inspection, deployed practitioners where needed, supervised the forming of societies, and thereby built up a profession where none had existed before. One must

*Presented at the 49th annual meeting of the New York State Veterinary Medical Society, Ithaca, N. Y., July 13-15, 1939, by the executive secretary of the American Veterinary Medical Association.

remember that the group of schools thus founded, without public sanction or support, was soon interspersed with meagerly supported successors which were to develop into the celebrated veterinary educational system which promises to take its place among the indispensable enterprises of American civilization.

The history of this evolution is punctuated with feature events, among which are:

a) The starting of our educational system in 1875 and the gradual increase in the length and scope of its curricula, corresponding to the advance of medical science;

b) the enactment of practice acts, which gave a legal name to our profession, and of the many laws and regulations governing our activities in our federal and state work;

c) the founding and development of the Bureau of Animal Industry, United States Department of Agriculture, which was to become a monument to the veterinary service of the world—a marvelous organization, commanded by veterinarians, famous for its scientific achievements and discipline;

d) the forming of livestock sanitary services under state veterinarians in all of the states which, by coöperating with the federal service, took the application of veterinary medicine to every nook of the country;

e) the gradual transfer of our educational system to the system of public instruction which was to give our country the veterinary colleges;

f) the founding of a veterinary corps for the army during and following the World War from nothing at the start to a fine, workable unit of the military service;

g) the addition of chairs on veterinary science in the agricultural colleges and the forming of the nationwide extension service, which in its formative period, still existing, has conflicted in certain respects with the objectives of the veterinary profession;

h) the elimination of equine transportation from the streets and highways, which turned us to the erstwhile neglected branches of veterinary medicine and thus gave birth to new fields of exploitation, the annual university conference, and the growth of organized veterinary medicine. In this respect, New York was the leader, and the late, lamented Veranus A. Moore the first sponsor; and

i) the phenomenal development of small animal medicine in the large cities, of which I shall speak later.

It is not my purpose here to relate what the profession has accomplished under the obstacles herein mentioned. Suffice to say that our effort, as J. S. Koen has ofttimes stated, has made the United States the safest place on earth to breed live stock, despite the handicaps that had to be removed at every step from our path of progress. All of this is a theme from a long historical essay—an essay which would draw public attention to man's dependence upon domestic animals in ample numbers and free from disease and to the unassailable fact that a nation's strength and security lies in the amplitude and health of its animal population. This fundamental fact is what has enabled our profession to grow in importance in spite of thoughtless neglect by the people through the years. It is the factor that enables the deans to get appropriations for their institutes and the practitioners to get laws governing their labors. Unless we can go on showing that our profession wisely managed is a public necessity, our fate as an accredited group will be beautifully thrown into the discard by the political and scientific circles which are just beginning to understand our motives—our *raison d'etre.*

SMALL ANIMAL MEDICINE A BRANCH OF THE VETERINARY PROFESSION—NOT A SEPARATE ENTITY

What I am driving at is this: If the branches of our art devoted to the labor of furnishing food and clothing to our increasing population find it difficult to bring public attention to the indispensable nature of their work, what would be the fate of the small animal branch without the other branches to uphold its dignity among the other professions?

From a long experience in the department of registration and education in the

State of Illinois, I have observed that legislatures, judges and juries look askance at the small animal branch, doubting that our practice act applies to pet animals. This being fact, not theory, my invocation to the small animal branch is to look beyond its beautiful surgeries and reception rooms in adjudicating its status and to seek closer and closer relations to the parent profession, rather than to set itself apart as an independent specialty.

It is supremely easy for frail man to get pretty egotistical from the mental effect of glamorous surroundings, if he does not look beyond his immediate environment. What a privilege it is to be rated as a part of such a tremendous undertaking as the eradication of bovine tuberculosis, Bang's disease, Texas fever, blackleg and hog cholera, not to mention the public health side of veterinary medicine! These are the factors which are making our profession. The small animal practitioner who does not stop to survey the veterinary service from the Pacific Coast, the Intermountain Region, the great ranch country, the famous Corn Belt, and the small farm areas to the Atlantic, lacks a true understanding of the profession to which he belongs, and his attitude toward these things is going to fix his place in veterinary medicine of the future.

Unethical Practices of Small Animal Branch Laid to Its Rapid, Undisciplined Growth

The small animal branch is young—very young—and it developed too rapidly to keep its personnel disciplined under the code of ethics which the large animal and sanitary branches developed with difficulty through the decades. The great display advertisements in telephone directories are responsible for incalculable injury to the profession. They set the small animal branch apart from its more ethical large animal colleagues, who with few exceptions have obeyed the common laws of professional conduct during days when quacks outnumbered graduates five to one, when advertising might have been used to attract public attention to the difference between quackery and scientific medicine.

I mention this because it is one of the great problems confronting the American Veterinary Medical Association, a problem that has grown with the rapidity with which its urban members have gone from large to small animal practice. Competition in this new field seems to have overlooked the teachings of the forefathers, who entertained no compromise in this respect and, as a consequence, did not succeed in building up the profession of which we are now proud and which we are striving to keep on the straight but narrow path of conventional conduct.

Commercialism and unethical behavior can not for long hide their faces behind science and beautiful hospitals, and win public approval and support. The various units of the service we here represent could not be maintained exclusively for the small animal branch, which has arrived at a point in the history of veterinary medicine in this country where it must choose between degenerating into a sort of glorified beauty parlors or joining wholeheartedly, by precept and example, in lifting the entire profession to the higher plane it seeks to attain.

Various Advertisements Mentioned

Small animal medicine, through the sentimental nature of its work, is in a position to build or destroy—build up by remaining close to the other branches, or destroy itself by standing apart as an independent specialty. I have before me, a number out of many hundred, intolerable advertisements. Let me read several picked at random:

> Male cats altered, $1.00; female cats spayed, $2.50. Dr. Doe, 10 X street.
> Plucking and clipping any sized dog, $2.50. Dr. X, etc."
> Female dogs, during April only, spayed for $2.50. Dr. B, 240 A street, etc.
> Permanent distemper inoculations, Dunkin—Laidlaw Improved, 3 injections, $5.50.

Unfortunately, the violators are graduates not of the old private schools but of the classical colleges of this hour. This makes me wonder in what direction the vet-

erinary profession is traveling. It certainly inspires the old timers to enlarge and fortify the associations—state and national—to a point where they will be able to take stern disciplinary action against behavior that is reducing the practice of veterinary medicine below the level of a common trade. While history can not be intelligently surveyed until a certain stretch of time has elapsed, here seems to be a trend that can be appraised at full value right now. In a profession unethical practices lead only in one direction—downward. There is no alternative. The whole structure of the veterinary service is threatened by these gruesome tactics.

A. V. M. A. MUST ACT TO RULE OUT UNETHICAL PRACTICES

You are going to ask why the A. V. M. A. does not take some action against these men. Well, don't be too sure that it is not going to do so. The national association is traditionally slow in handling such matters but it is also traditionally sure of its ground. When it has acted during its 76 years, it has never had to reverse itself. The A. V. M. A. is not unaware that small animal medicine is an important branch of the service that is groping its way upward against odds, that has not yet established an approved system of ethics for its members; nor is it unaware of the fine scientific technicians belonging to it. In other words, the national association is tolerant but firm and, as a consequence, action in this connection is not far ahead. We are beginning to believe that if we discipline some 500 or 600 advertising doctors, we would certainly gain a lot of respect from those who desire to travel the straight road and probably add twice that many members who disapprove advertising.

A good way to progress in this connection is for the practitioners of cities to come to an understanding as to advertising in telephone directories, as several such groups have already done and, thus, made a clear distinction between the non-advertising and advertising doctors. It is surprising how quickly the dog-owning public grasps the idea. Who in looking for the name of a physician, or lawyer, or dentist in a telephone directory would select an advertiser?

Secretary Fehr requested me to talk on diseases of small animals, not knowing that telling you men of that demesne how to treat small animals would be quite a strange presumption. Small animal medicine was always a sideline with me and I have not practiced for 22 years. Anything I could bring to you on this subject would be what I have "read in the newspapers," as Will Rogers used to say. So, I chose to change the subject to "Diseases of the Small Animal Branch"—a branch of "pathology" my present position compels me to study. Having been the president of the national association who installed a small animal section into that society, my interest in the subject should be clear. That is, I should not have to make apologies for making suggestions on how that branch should deport itself and how it should be dovetailed into the work of organized veterinary medicine to the best advantage of the American people.

SCIENTIFIC CONTRIBUTIONS OF SMALL ANIMAL BRANCH LAUDED

The veterinary profession owes a lot to the small animal practitioners for the refinement they have brought into clinical work. They have revolutionized the clinic in their own field and, by example, have brought neglected diagnostic and surgical methods to the attention of the large animal branch. The large clinics carried out by Iowa and Illinois veterinarians—singled out because I am familiar with them—are testimonials to the small animal branch, which gave us hematology, chemistry and microscopy on the one hand and Listerian methods on the other in the handling of ailing animals. What a pity it would be if the branch that has given us so much should prove to be the one that threw aside the hallowed code which, we believe, must forever be obeyed to win and retain public approval of our profession.

In order to compensate for not dwelling upon diseases and treatments, as Secretary Fehr obviously intended I should, may I

digress from my theme to mention the subjects which seem to be of current interest, namely:

a) The scientific dosage of sulfanilamide and its true indications, its mode of action, and its contraindications;

b) the indications and uses of nicotinic acid and the prodromal signs of various avitaminoses;

c) the use of vitamin E in reproductive disorders and the determination of vitamin E deficiency;

d) iodine deficiency in small animal practice;

e) the nutritive value of prepared foods;

f) the true indications of tonsillectomy;

g) the immunizing value of rabies vaccine in single doses;

h) canine and feline tuberculosis; and

i) sulfapyridine in canine pneumonia.

If my paper falls short of meeting the desires of this audience, here are a number of topics suggested for your deliberation. My personal opinions on any of them would not be taken seriously east of the Alleghanies.

Nicotinic Acid—Sulfanilamide

The last half of this decade goes down in medical history as the date when two great discoveries were made in pharmacology. The one is sulfanilamide, a dye, brought into the practice of medicine by Domagk of Germany; the other is nicotinic acid, fraction of vitamin B, signalized by our own Elvehjem of the University of Wisconsin. Except that each of these two drugs actually cures a serious disease, they have nothing in common. They are derivatives of different kingdoms and they attack disease by different strategy. The one strikes directly at pathogenic organisms, the other cures by correcting somatic nutrition.

Sulfanilamide was the dream of Paul Ehrlich, who, in the early studies of bacteriology, sought the discovery of chemical agents having elective action (affinity) against invading microbes but which would not seriously inconvenience the host. Nicotinic acid was the hope of Elie Metchnikopf, who sought means of propping up the cellular defenses which would unarm the microbic invaders of higher life. Pasteur was skeptical about these two "schools of thought." To him, killing of his invulnerable microbes nesting in or among the delicate cells of the body was a forlorn hope of internal medicine. As to the philosophy of his learned contemporary, who visioned armies of cells coming to the rescue of an invaded locale, he was noncommittal, content with his chosen field of antigenic therapy.

Ehrlich's idea led to the curing of spirocetoses with arsenicals and to that of bacterial diseases with sulfanilamide, while Metchnikopf's turned the mind to the study of natural defenses capable of flanking the invading enemy. Thus, two classes of antimicrobic remedies were created: the microbicide on the one hand and the builder of body vigor on the other. Sulfanilamide belongs to the first class and nicotinic acid to the second. Their dynamics should not be confused.

The victory of sufanilamide over certain microbic diseases is too well known to need rehearsing here. It is a chemical deadly to erstwhile unmanageable pathogens—a cure for heretofore incurable infections. As a victorious chemical it has no peer, not even in the salvarsan of Ehrlich or the calisaya of the ancient Peruvian, judging from the wealth of favorable reports contained in the medical literature of this hour.

The victory of nicotinic acid is of a different order. It cures disease by removing the cause. It concerns nutrition, not microbe killing, and from that token reopens the broad field of causation which has lain but half-explored despite the suggestive work of Eijkman, proponent of the vitamin theory, which led to the discovery of vitamin B and ultimately to this divisible component which cures as if by magic the human pellagrin and the canine victim of blacktongue, without possessing any microbicide action whatsoever.

The founding of formal education in animal medicine during the 18th century led to the forming and the wealth of the great nations of this period.

A Growing Yeast Medium for the Cultivation of Hemophilic Bacilli*

By JOHN P. DELAPLANE, D. V. M., and H. O. STUART

Kingston, R. I.

THE POOR growth yield of *Haemophilus gallinarum* in chicken and other blood media and the necessity of making frequent transfers to enable successful cultivation suggests the value of a more practical blood-free medium. A blood-free medium dispenses with the need and overcomes the inconveniences of obtaining suitable sterile blood for maintaining stock cultures.

The growth requirements of the fowl coryza organisms have been studied by Schalm and Beach,[1] Kessens[2] and Delaplane, Erwin, and Stuart.[3] These studies have indicated that certain factors found in blood, but which may be supplied from other sources, are necessary for growth.

Delaplane, Erwin, and Stuart,[3] studying a yeast extract, found that it furnished certain of the factors necessary for growth. Delaplane and Stuart[4] used a blood-free medium which utilizes growing yeast to furnish the growth factors for *H. gallinarum* and *influenzae*. This medium resulted in a growth of *H. gallinarum* heavier than that obtained with a chicken-blood medium.

The morphological differences between yeast cells and *H. gallinarum* make it possible to determine growth by using stained smears for microscopic examination. If pure cultures of *H. gallinarum* are desired, they can be readily isolated from yeast by plating on chicken-blood agar and grown in a chicken-blood medium.

DISCUSSION

The pure cultures of yeast used for the studies were isolated from a commercial dried yeast. Extracts of this commercial yeast furnished certain of the factors necessary for growth of *H. gallinarum*.

The yeast was first grown in sterile Difco dehydrated phenol-red maltose or dextrose solutions (Difco phenol-red maltose or dextrose, 13 grams; H_2O, 1,000 cc.; NaCl, 8 grains) for several days at room temperature. The acidity resulting from the yeast growth was made alkaline with NaOH just prior to inoculation with cultures of *H. gallinarum*. *H. gallinarum* grew in this medium but the results were not uniformly good.

In using some of the yeast-inoculated sugar solutions, as just described, at the base of nutrient agar slants, it was noted that the pH of the medium tended to become alkaline near the surface of the solution, even though NaOH was not used to readjust the reaction. This was desirable as far as growth of *H. gallinarum* was concerned, and eliminated the necessity of readjusting the pH. Difco dehydrated phenol-red maltose and dextrose were separately added to Difco nutrient agar (Difco nutrient agar, 23 grams; Difco phenol-red maltose or dextrose, 10 grams; salt, 8 grams; H_2O, 1,000 cc.), tubed and sterilized for use to determine whether the growth of yeast would bring about the change of pH on a solid medium. Slants of the tubed media were inoculated with yeast and incubated at 37° C. A moderate growth of yeast resulted, and the pH of the medium became alkaline, as indicated by the phenol-red.

To start the mixed cultures, sterile citrated chicken blood was added to the base of the slanted media and inoculated with *H. gallinarum*, following which the exposed surface of the slant was inoculated with yeast. There was a good growth of the organism at the surface of the blood where it contacted the yeast. Subcultures were made from the initial mixed cultures by inoculating the surface of the slants of the experimental blood-free media with a large

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loopful of the yeast and *H. gallinarum*. A small amount of sterile normal salt solution was added to the base of the slant to prevent drying.

The organism continued to grow with the yeast in much greater quantities, as indicated by staining, than could be obtained from chicken blood. Transfers were made at weekly intervals by seeding the medium in a manner already described. The addition of potato extract to the medium seemed to improve it and did not prevent it from becoming alkaline following growth of the yeast. The completed medium had the following composition: Difco nutrient agar, 23 grams; Difco phenol-red maltose or dextrose, 10 grams; salt, 8 grams; added to 1,000 cc. of broth or H_2O in which 400 grams of potato had been cooked.

Cultures of *H. gallinarum* have been cultivated continuously in the growing yeast medium for over a year, transfers being made at weekly intervals. Cultures of *H. influenzae* also have been grown on this type of medium. The growing yeast medium has the advantage over blood in that the medium can be sterilized by heat and can be utilized by simply inoculating the surface of the slants with the mixture of yeast and hemophilic bacilli. If blood is used, several steps requiring asepsis are necessary to obtain sterile blood suitable for cultivation of the organisms.

The two organisms growing together produce a sticky, mucoid-like growth. Pure yeast cultures lack this mucoid characteristic. *H. influenzae* and yeast also show this mucoid characteristic, but not to the same extent.

The results of chicken inoculations with *H. gallinarum* grown with yeast indicate that the virulence of the organism is not reduced. Some evidence is available which indicates that the virulence was retained better in the yeast-grown cultures as compared to the same organism grown in blood.

Some strains of *H. gallinarum* seem to grow better than others in the yeast medium. Whether this is dependent upon the length of time they were cultivated in the laboratory prior to using yeast is not known, but we did observe that strains which had been cultivated for a long time grew best.

A smooth strain of *H. influenzae* grew much better than a rough type of this organism. Whether this might explain differences noted with *H. gallinarum* has not been determined.

The success of the medium is dependent upon the medium becoming alkaline by the action of yeast. The medium in tubes which were sealed or stoppered did not become alkaline and very little growth of *H. gallinarum* or *influenzae* occurred.

Studies to determine the nutrient responsible for the pH change of the medium would indicate beef extract, because if peptone were substituted for beef extract, the medium would become and remain acid.

The medium has been prepared by using the same proportions of the various ingredients, Bacto phenol-red maltose or dextrose and Difco nutrient agar, with equal success. The phenol-red indicator can be omitted, if desired, without affecting the results. These preparations were used and studied because of earlier observations of the reactions which occurred following their use.

Whether the organisms grown in this manner would be suitable for serological studies has not been determined, although some preliminary tests in this respect would seem to warrant further studies. Suspensions of the mixed cultures indicate that the yeast settles to the bottom of tubes much quicker than the hemophilic organisms upon standing or centrifuging, and thus they may be partially separated

The use of live or attenuated vaccines, prepared from yeast-grown organisms, to stimulate immunity in chickens against infectious coryza was found to offer no encouragement in this respect.

Whether other types of yeast will give as good or better results has not been determined.

SUMMARY

A blood-free medium, utilizing live, growing yeast to furnish the necessary

growth factors for *Haemophilus gallinarum* and *influenzae* is described.

The medium results in a better growth of the organisms than that obtained with a chicken-blood medium.

Vaccines prepared from the yeast-grown cultures do not offer any encouragement for vaccinating chickens against infectious coryza.

The value of the yeast medium for growing hemophilic organisms for other studies is suggested.

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Worms and Bacteria in Fowl Paralysis

In 1938, Ernest Gray, M.R.C.V.S., F.R.M.S., of the East Anglian Institute of Agriculture (England) reported that streptococci and staphylococci and, in one case, *Bacillus pyocyaneus* had been found in the nerves of fowl affected with fowl paralysis. In a later observation on 58 affected fowl examined post mortem, a heavy infestation of coccidia was found in 49, tapeworms in five, roundworms in two, and a nephritis in one. In 37 of the cases affected with coccidiosis, coccoid bodies were found in the affected nerves and also in the spinal fluid. Repeatedly, the disease was found associated with coccidiosis. Birds experimentally infected with coccidia together with subcultures of *Streptococcus viridans* and *B. pyocyaneus* developed an edematous condition of the sciatics, coccidial enteritis, and nephritis. Staphylococci, Gram-negative bacilli, and Gram-positive cocci were isolated in the sciatic nerves. *(The Veterinary Record, li, June 17, 1939, p. 754.)*

Materia Aphrodisia

Spanish Fly, although belonging to the spineless creatures of the earth, is an animal of considerable importance. It was prescribed by Hippocrates as an aphrodisiac and is still used for that purpose today, according to Allen Wright (*The American Mercury*, July, 1939). "Like yohimbine," says the author, "cantharides is successfully used upon animals, usually the smaller ones, such as rams, dogs and pigs. Among the other excitants of the sexual impulse named by the author are arsenic, strychnine and wheat germ oil. Of the latter he says: "Within the last few years wheat germ oil has come into prominence as an aphrodisiac drug. Obtained by pressure from the tiny germ of the wheat grain, the drug contains a high concentration of vitamin E, the reproductive vitamin. Males (human) and animals deprived of this vitamin invariably become sterile. The oil was first used as a remedy for female frigidity. Later, it was reported to have definite value to restore sexual potency in the male."

Animals and Air Raids

The Royal Society for the Prevention of Cruelty to Animals is all "het up" over the failure of the government to provide protection for animals in the case of air raids from the mainland. But, says Wm. Scott in *The Veterinary Record*, that detail should be left to the Royal College of Veterinary Surgeons in times of peace and to the veterinary corps when war comes along. The R.S.P.C.A. advertises its activities but the veterinarians "hide their modesty under a bushel," Scott admonishes.

Though there is no comparable situation to arouse interest in this country, the circumstance is mentioned here to infer that veterinarians everywhere are not good advertisers.

Milk and cheese are not constipating. They are merely digested and absorbed without leaving the amount of residue (bulk) required to stimulate bowel action.

Vesicular Dermatitis in Chickens

By H. A. HOFFMAN,* D.V.M.

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A DISEASE characterized in the early stages by vesicles and, later, by amber-colored crusts, or scabs, on the comb, wattles, face, feet, and shanks was observed in a flock of White Leghorn hens during April, 1937. The entire adult chicken population was affected. This comprised 2,600 hens in five separate pens. Daily egg production dropped from 1,600 to 700 during the course of the disease. The owner estimated a death loss of 10 per cent. This case is identified as Ac. 384.

A flock of several hundred 10-week-old pullets on the same ranch failed to develop lesions. However, the owner reported mild lesions on the comb of a few cockerels associated with the pullets.

During the course of this investigation, birds from other flocks showing some of the same lesions were examined. These included two cases in which lesions appeared on the unfeathered portions of the skin, while other cases showed inflammations of the joints of the feet.

Newsom and Feldman[1] described a disease of chicks that was characterized by blisters between the toes, on the feet, and, sometimes, on the unfeathered portions of the head. The symptoms and lesions were very severe, with a mortality of 90 to 100 per cent in affected flocks. They stated:

> Older chickens are not nearly so frequently affected, but when the disease does attack them, it produces similar conditions of the feet and shows considerable swelling around the eyes, with rather extreme redness. Vesicles have not been noted in hens, it requiring from one to two weeks in most instances for the disease to prove fatal.

These writers failed to demonstrate the cause of the condition that they observed. They indicated, however, that evidence pointed to some association of the condition with ranging chickens on unbroken scd.

Figs. 1 and 2. Ac. 384. Scabs on comb, wattles, eyelids, and around the beak.

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SYMPTOMS AND LESIONS

The onset of the disease was rapid. Vesicles appeared on the comb, wattles, other unfeathered portions of the head, the feet and hocks. The content of the vesicles was milky and sometimes greenish in appearance. After a few days the vesicles were replaced by thick, amber-colored crusts or scabs. (See figures 1-3.) In many birds, the eyelids stuck together.

As the disease progressed, new vesicles appeared and in some birds the lesions on the feet and legs involved the entire skin from the feet to the hocks until the entire scaly portion of the skin of the shank was loosened.

The crusts on the combs were firmly attached and difficult to remove. If removed forcibly, the underlying tissues appeared smooth and deep pink. The amber-colored crusts on the head and feet persisted for at least three weeks. During this period, the birds' appetites were poor. Examination of the internal organs failed to reveal lesions of any type.

The original outbreak lasted for approximately four weeks. Thirty days after the first cases were observed most of the birds had partially or completely recovered. Five weeks after the start of the original outbreak new symptoms appeared in some of the birds that had recovered from the first attack. Birds previously affected were identified by scars of the original lesions, especially by interruption in the growth of the beak and an offset in the horny structure at the injunction of the older and new growth of this organ (fig. 4). Several birds were taken to the laboratory at the start of the original outbreak. These were held under observation for several months. It is significant that fresh lesions developed in some of these birds at the same time that the disease recurred in the flock on the ranch.

The character of the symptoms and lesions suggested the possibility of an infectious disease. Cultural examination of a number of the affected birds yielded an unpigmented staphylococcus. In some instances, this organism was isolated in pure culture. In other cases it was associated with various bacteria and moulds. The possibility of a virus disease was considered. Unfortunately, it was not practicable to investigate this in an experimental manner.

EXPERIMENTAL PROCEDURE

Attempts were made to transfer the disease to healthy chickens by pen contact, force feeding of lesions and cultures, and the inoculation of blood, lesions from the affected parts, or cultures of the coccus isolated from field cases.

A young cockerel was placed in a wire cage, 2 feet square, with two hens showing active lesions of the disease. Neither symptoms nor lesions developed as a result of this exposure.

Blood from an affected hen was injected intraperitoneally into two chickens. The results of this test were negative. Material from the lesions of the skin of affected chickens was fed in liberal quantities to four chickens, with negative results. Exudate from the skin of affected birds was rubbed into the scarified skin of the comb, feet, or wattles of twelve chickens. Of these, five inoculations yielded negative results. In seven of the birds small vesicles appeared promptly. These were followed by crusts, which persisted for some time.

Pure cultures of the cocci recovered from the affected parts were rubbed into the scarified skin of the head and the feet of eight birds. The results of three of these inoculations were negative. The remaining five developed vesicles followed by scabs.

The lesions produced in birds inoculated with material from field cases, as well as those inoculated with cultures, had the same characteristics as the lesions in natural field cases. There was, however, little tendency for the lesions in inoculated birds to spread beyond the point of inoculation. One bird, inoculated in the web of the foot, developed a swelling of the joints that persisted for some weeks.

During the course of this investigation, birds from a breeding flock of White Leghorns, identified as Ac. 513, were brought to the laboratory for examination. The owner reported the repeated occurrence of lameness with swelling in the joints of the feet. Lesions in the region of the head were not reported. The association of staphylo-

cocci with arthritis in chickens and turkeys has been reported by several authors.[2-5] Cultural examination of the lesions of the feet yielded a coccus that was morphologically similar to that recovered from Ac. 384.

Exudate from the lesions in the feet of a bird from this flock was rubbed into the scarified comb and scarified skin beneath the wing of a healthy bird. No lesions developed on the comb. A moist, yellow scab formed at the point of inoculation beneath the wing. Later tests, in which the skin was scarified without introducing infective material, tended to discount the significance of the results of this inoculation. A hen was inoculated in a similar manner, pure cultures of the cocci isolated from this case being used. After 24 hours small, white blisters appeared on the comb in the scarified area. A moist, yellow scab appeared beneath the wing. Further tests were not made with this material.

Another case, Ac. 494, was brought to the laboratory for examination. There were two 3-week-old chicks. According to the history given, 100 of these chicks were running with hens. About 15 or 20 had scabs on the feet. The hens were not affected. The chicks examined showed the following lesions: Chick 1—blister on comb, swelling of the feet, and edema of the feet and shanks; chick 2—small scab on comb and swelling of the feet and shanks. Cultures revealed a Gram-positive staphylococcus.

A young cockerel was inoculated with material from Ac. 494 by rubbing exudate from the feet and comb into scarified areas on both sides of the comb, on the web of the right foot, and beneath the right wing. The following day blisters were observed on the comb, wing, and web of the foot. Material from the right wing was rubbed into scarified skin beneath the left wing of the same bird. The following day blisters appeared in the area of this inoculation. A coccus was recovered in culture from both wings and from the comb. Another bird was inoculated on the same date, in the same manner, in the comb and web of the right foot. Comb lesions failed to develop but a slight swelling of the joints of the inoculated foot appeared after four days.

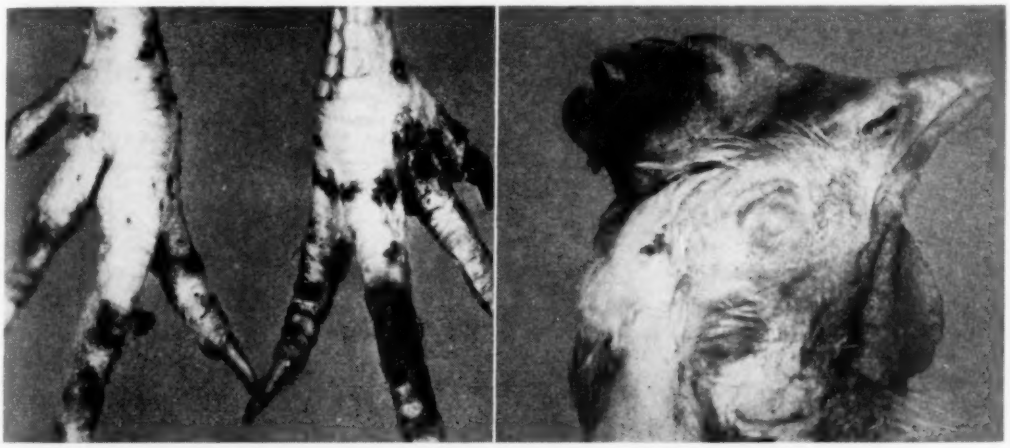

Fig. 3 (left). Ac. 384. Scabs on feet and shanks resulting from vesicle formations in the skin.
Fig. 4 (right). Ac. 384. Scabs on comb as the result of a second attack approximately five weeks following the first attack.

Material from the wing of the first bird was inoculated into the right wing of the second bird. After 24 hours blisters appeared. A third bird was inoculated in the scarified areas of the comb, foot and wing with cultures of a coccus recovered from the original field case, Ac. 494. Blisters appeared beneath the wing but not on the comb or feet.

Another case, Ac. 1880, represents a chick 7 weeks old. The tissues of both feet were swollen and inflamed (fig. 5). The inflammation extended upward along the

tendons almost to the hock of one foot. The wattles were swollen. The owner said that several of his chicks were affected.

Cultures from the wattles yielded a small coccus, while cultures from the feet were mixed. Inoculation tests were not attempted in this case.

The character of the lesions found in field cases, especially Ac. 384, suggested a resemblance to impetigo in man. Through the courtesy of a physician, a swab from a human case of impetigo was obtained for study. Cultural examination revealed an organism very similar in appearance to that recovered from all of the cases previously mentioned.

Fig. 5. Ac. 1880. Vesicles on the feet of a 7-week-old chick.

Exudate from the impetigo case was rubbed into a scarified area of the comb of a hen. After 48 hours several small blisters appeared at the point of inoculation. These lesions were followed by yellowish crusts or scabs. Another hen was inoculated in scarified areas in the comb, foot, and wing with cultures of the coccus obtained from the impetigo case. Extensive moist scabs appeared at all points of inoculation. Vesicles were not noted.

SUMMARY

An acute outbreak in hens of vesicular dermatitis that involved the unfeathered skin of the head, feet and shanks is reported. One attack of the disease failed to confer an immunity, as evidenced by the fact that the disease recurred in the same individuals approximately five weeks following the onset of the first symptoms.

Cultural examinations of affected birds consistently yielded staphylococci from lesions on the head and feet and, in one instance, from the liver of affected birds. Other types of organisms, apparently contaminants, were found in some of the cultures.

Exudate from the lesions on affected birds, as well as cultures of the staphylococcus, when rubbed into scarified skin of healthy birds resulted in vesicle formations in twelve out of 20 inoculations made. Pen contact, feeding tests and subcutaneous inoculations were negative. The mild type of lesions produced by inoculation tests and the failure of these lesions to spread after the manner observed in natural cases preclude definite conclusions with respect to the etiological significance of the organisms studied.

Comparative studies were made with staphylococci obtained from cases of arthritis in chickens and from a case of impetigo in man. Inoculations with these cultures into scarified skin of healthy chickens resulted in lesions indistinguishable from those obtained with cultures from the acute vesicular dermatitis case.

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Henrietta, a White Minorca hen owned by Frank Peglow of Elgin, Ill., laid an egg in the henhouse a short time ago. Such an act would normally be unworthy of annotation, but it happens that Henrietta is 20 years old.

Because of the great volume of material carried out of hens with the eggs they lay, depletion of their bodies of essential elements is greater than in domestic mammals. To maintain somatic normalcy in laying hens, the intake must match the output precisely; hence the importance of diet in poultry husbandry.

The Viability and Immunizing Value of Egg-Propagated Laryngotracheitis Virus*

By F. R. BEAUDETTE, D.V.M., and C. B. HUDSON

New Brunswick, N. J.

IN THE PRODUCTION of laryngotracheitis virus for vaccination, tracheal exudates are removed from artificially produced cases of the disease, dried, ground, and stored in stoppered or in evacuated, hermetically sealed ampuls. At the time of vaccination 20 mg. of the dried virus is mixed with 1 cc. of a 50 per cent glycerin solution, which gives an amount sufficient for 50 vaccinations.

In our experiments some years ago, virus dried at room temperature gave good results if used within a short time. Drying in the incubator also gave a suitable product for use within a limited period,[1] but the present method is to dry the virus under vacuum over a desiccating agent.[2] Such a virus maintains its potency long enough to be sufficiently active within the expiration date set by most commercial laboratories. However, if potency is not maintained, the vaccine gives a low percentage of "takes." In this case the majority of birds that fail to show a readable "take" are not immunized and will usually contract the disease about the tenth day after vaccination. In order to insure a potent vaccine, some have attempted to use suspensions of freshly harvested virus, but this merely increases the possibility of spreading other diseases through the medium of contaminated vaccine.

Naturally produced virus is of necessity contaminated with the usual flora of the upper respiratory tract. To what extent bacterial activity reduces the potency of natural virus up to the time of complete desiccation has not been determined, but it may be a factor. Of far greater importance is the fact that if great care is not used in the selection of birds for virus production, pathogenic bacteria and viruses may contaminate the exudates. But, a pathogenic contaminant to be of importance must survive the desiccation and then be able to infect directly by intracloacal inoculation or indirectly by escaping from the cloaca and reëntering through its natural channel.

The common occurrence of the fowl cholera organism, *Pasteurella avicida*, in the nasal cavity has been well established.[3] Mild infections due to the coryza bacillus, *Haemophilus gallinarum*, could easily be overlooked, and in rare cases a fowl might be a carrier of a hemolytic streptococcus.[4] As regards possible virus contaminants, fowl pox and infectious bronchitis must be seriously considered.

Although Doyle and Minett[5] have shown that after active lesions disappear a bird no longer carries pox virus, the fact remains that a small lesion might easily escape notice at the time of artificial inoculation for virus production or the fowl may be in the incubation stages of the disease. In the latter event cutaneous lesions would be easily identified at the time of harvest but differentiation of the mucous membrane lesions would offer a real problem. Since older birds are used for virus production and since bronchitis is always a mild disease in them, an infection with this virus might easily escape notice. Finally, the virus of leukemia is known to occur in the blood and apparently the causative agent of lymphomatosis is also to be found there.[6]

Although fowl plague and New Castle disease are not known to exist in the United States at the present time, plague has made two appearances in recent years. In both diseases tracheal material would be rich in virus. Moreover, there is good reason to suppose that vaccination against laryngotracheitis may be practiced in countries where it has been diagnosed recently any-

*Presented at the 74th annual meeting of the A. V. M. A., Omaha, Neb., August 16-20, 1937. From the New Jersey Agricultural Experiment Station.

where, in some cases at least, plague or New Castle disease, or both, are known to exist.

Assuming that an exudate is contaminated with any of the disease-producing agents mentioned, the question arises as to which of these would survive the drying process. From what is known of the coryza bacillus it is safe to say that it would not withstand drying. The streptococcus infection encountered in New Jersey would not stand drying for any length of time, but the cholera organism might remain viable. Of the possible virus contaminants all of them with the exception of lymphomatosis would certainly withstand desiccation. Recently, we found that dried bronchitis virus was still potent after nine months.

Finally, assuming that the exudate is contaminated and that the contaminant was preserved by the drying process, it becomes a question of which of these is capable of infecting a fowl by intracloacal inoculation. Nelson[7] has shown that this mode of inoculation with the coryza bacillus will not cause infection. The respiratory tract is the natural portal of entry of the cholera bacillus and the streptococcus. If these organisms fail to infect by intracloacal inoculation, they might escape from the vent and reënter the body through the natural portal of infection.

In one of our tests approximately 100 birds were inoculated in the vent with pigeon-pox virus and not one showed infection, but fowl-pox virus would likely produce infection. Todd[8] has shown that intracloacal inoculation with plague virus gives uniform infection, but New Castle virus given by this means would probably be less infective. Nothing is known of the natural portal of entry of leukemic virus or of lymphomatosis, but since the former disease can be induced by several means of inoculation, intracloacal inoculation might give occasional positive results. It is evident that in any event the use of freshly harvested exudate is hazardous and that the practice of using "homemade" vaccines in cases of emergency, as recommended by some, is to be condemned.

However, commercial laboratories are required to make tests for contamination on every lot before it is released for distribution. And, while these tests appear to be adequate for the usual possible contaminants, the advantages of a known pure virus are clearly evident. Such a virus can be produced on the chorioallantoic membrane of the developing chick embryo.

Some experiments along this line have already been made by Burnet[9] and Brandly.[10] In order to be of practical value it must be shown, first, that continued progagation in eggs does not alter the immunizing power of such virus, and, second, that egg virus dried will maintain its potency over a long period of time. In Burnet's[9] experiments various passages up to the 25th were tested for virulence by intratracheal inoculation. In these tests the virus was never dried, as would be necessary under commercial conditions; nor was it kept for any length of time, for the tests were said to have been made the following day. However, under the conditions of the tests, the virus was potent and the surviving fowls were immune to further intratracheal inoculation.

Brandly[10] tested virus after 35 passages and concluded that it showed no alteration or modification in infectivity for eggs or young chicks. Third passage virus dried for one month had lost none of its potency for eggs. However, Brandly makes a significant statement to the effect that virulence for cloacal tissue generally appeared to be somewhat lower than for tracheal mucosa. Since, in vaccination, virus must infect the cloacal mucosa, it seems to us that any test for potency must realize this method of inoculation rather than intratracheal inoculation.

In Brandly's four vaccination experiments, in which tracheal virus was compared with egg virus, no statement is made regarding the age of virus at the time of use. In some cases it was apparently used fresh and in other cases after being dried. In one case it appears to have been dried for a period of six weeks; older virus seems not to have been used. The virus was used at the rate of 10 or 100 mg. per cc. of diluent. In three experiments the birds were tested for immunity by intratracheal inoculation three to four weeks after vaccination. Since the percentage of immunes was lower in every case than the percentage

of "takes," it is clearly evident that some false positive reactions were recorded. In our very extensive experiments we have never found a bird susceptible after showing a definite "take." On the contrary, we usually get 97 per cent or more readable "takes" in routine vaccination, and the percentage of immunes actually runs higher than this.

From the experiments reported thus far it appears that continued propagation of the virus in eggs does not alter its immunizing properties, but data is still lacking on the keeping qualities of such virus. Consequently, our experiments were planned to throw some light on this latter point.

EXPERIMENTS

The first generation was initiated December 12, 1935, by the inoculation of six eggs with a Berkefeld V filtrate. One embryo died early and of the five remaining, three died on the fifth day and two on the sixth day of incubation. The membranes from one of the embryos dead on the sixth day were used to initiate the second generation. The detailed information of subsequent passages is given in table I. The virus is still being propagated but the experiments to be reported here concern only the first 25 generations. Table I indicates the generation, the date it was initiated, and the age of the eggs inoculated. As regards the inoculum, the membranes from a single egg were used and the number of days that that egg was incubated after inoculation is given and it is also stated whether the embryo was dead or alive at harvest.

If the membranes were not harvested as soon as death was determined, the length of time the egg was held in the refrigerator before harvest is indicated in the second column under "inoculum." With the exception of the 22nd generation, the membranes were held in a frozen state between inoculations for the length of time given in the third column under "inoculum." Early deaths, which are not considered, invariably took place in the first 24 hours after inoculation. The membranes for egg inoculation were ground in broth and the dose was approximately 0.6 cc., except in the 20th generations, in which the first three groups suffering a high early mortality received 0.8 cc. of a 1:4 dilution of the inoculum given in a dose of 0.2 cc. to the three remaining groups in this generation.

In all, nine lots of vaccine were made. Seven of these were made from tenth generation virus and differed only with respect to the age of the embryonated egg at the time of inoculation. Lot 1 was made from eggs 17 days old at the time of inoculation and lot 7 from eggs 11 days old at the time of inoculation. As indicated by table I, all membranes were harvested after four days of incubation, dried *in vacuo* for five days, and ground and stored in a desiccator in the refrigerator thereafter.

Membranes are extremely hard to grind after drying unless infection is quite generalized. Consequently, when the two lots of vaccine were made from 25th generation virus, lot 9 was ground before drying. Otherwise, these two lots were treated like the first seven. It should be added that the 25th generation consisted of three groups of eggs. The first was inoculated with a 1:5 dilution in saline, the second with a 1:5 dilution in broth, and the third with a concentrated broth suspension. There appeared to be no difference in the extent of infection in the three groups.

TITRATION

The titrations were by no means calculated to determine the limit of activity. Since all commercial laryngotracheitis vaccines, to our knowledge, allow 20 mg. of dried virus per cc. of diluent, there was no object in using more concentrated suspensions in the titrations. Hence, for the first seven lots a suspension containing 20 mg. of virus per cc. of 50 per cent glycerin was made and, from this, other dilutions containing dried virus at the rate of 10, 5, and 1 mg., respectively, were made. For the titration of lots 8 and 9 the most concentrated suspension contained 5 mg. of dried virus per cc., and others contained 2.5, 1.25, and 0.625 mg., respectively.

The experimental chickens had been grown from the day-old stage in an electrically heated and ventilated battery room in a room within another room, and were cared for by a person who never came in

TABLE I—Detailed results of egg passages to the 25th generation.

Generation	Date of Inoculation	Age of Eggs When Inoculated	Inoculum			Number of Eggs Inoculated	Number Early Dead	Number Remaining	Results						
			Days Egg Incubated	Days in Egg After Death	Days Held in Freezer				3rd	4th	5th	6th	7th	8th	9th
2	12–20–35	11	6D	0	2	9	0	9				9D			
3	12–26–35	13	6D	0	0	6	1	5		2D					
										3A					
4	12–30–35	9	4A	0	0	11	2	9	5D	4A					
4a	1– 6–36	11	4A	0	7	9	1	8			1D	1D	3D	1D	2D
		11				5	0	5		5D					
5	1–13–36	10	7D	0	0	4	0	4		4D					
		9				2	0	2		1D	1D				
6	1–17–36	12	4D	0	0	2	0	2	2D						
7	1–24–36	10	3D	4	0	10	1	9			3D	6D			
		11				3	0	3			3D				
8	1–31–36	13	5D	2	0	4	0	4		1D	3D				
		15				5	0	5		1D			4D		
8a	2–29–36	11	6D	1	29	3	0	3				1D	1D		
													1A		
9	2– 5–36	12	5D	0	0	6	0	6	2D	3D	1D				
9a	2–10–36	13	4D	6	0	8	1	7			1D				
											6A				
		17				8	1	7		1D					
										6A					
		16				6	0	6		6A					
		15				9	1	8		5D					
										3A					
10	2–15–36	14	5A	0	0	8	2	6		4D					
										2A					
		13				9	0	9	1D	6D					
										2A					
		12				9	1	8	1D	7D					
		11				12	0	12	1D	9D					
										2A					
11	2–19–36	11	4D	0	0	2	0	2		2D					
12	2–24–36	13	4D	1	0	1	0	1			1D				
13	2–29–36	11	5D	0	0	3	0	3		3D					
14	3– 5–36	14	4D	1	0	3	0	3		1D		2D			
15	4– 3–36	10	6D	3	20	5	0	5		1D*		1D	1A		
												2A			
16	4–13–36	11	6A	0	4	3	0	3			3A				
17	4–22–36	11	5A	0	4	4	1	3			1D				
											2A				
18	5– 2–36	10	5D	0	5	3	0	3				3D			
19	5–11–36	12	6D	0	3	8	1	7			4D				
											3A				
19a	5–13–36	9	6D	0	5	3	0	3		1D*	2A				
		13				8	5	3	3A						
		12				9	2	7	7A						
20	5–25–36	11	5A	0	9	9	4	5	5A						
		13				8	0	8	8A						
		12				9	1	8	8A						
		11				8	1	7	7A						

(*Continued on next page*)

(1) Virus held in a dried state.
* These are the only eggs which failed to show an anatomical evidence of infection.
D = Embryo dead.
A = Embryo alive when egg was opened.

TABLE I—Continued.

20a	6–27–36	11	5A	0	42	6	0	6	1A*		5A				
21	6–22–36	12	3A	0	25	7	1	6	1D	1D					
										4A					
21a	7–13–36	10	5A	0	10	4	0	4		4A					
22	10–26–36	12	4A	0	101	5	3	2			1D				
											1A				
23	11–26–36	9	5D	0	16	5	2	3			3A				
24	11–30–36	11	5A	0	9	10	2	8			1D				
											7A				
		10				7	0	7			1D	4D			
												2A			
25	12–15–36	10	5A	0	10	7	1	6				2D			
												4A			
		10				6	1	5				3D			
												2A			

TABLE II—Results of nine titrations of lots 1 to 7.

Age of Virus (Days)		6	35	66	118	159	264	296	331	421
Titrations		1	2	3	4	5	6	7	8	9
Lot	Mg. Virus per cc.	Feb. 25 1936	Mar. 25 1936	April 25 1936	June 16 1936	July 27 1936	Nov. 9 1936	Dec. 11 1936	Jan. 15 1937	April 15 1937
1	1	xxx	xxx	xxx	xxx	xxxx	xxx	xxxx	xxx	xxx
	5	xxx	xxx	xxx	xxxx	xxx	xxx	xx	xxx	xx
	10	xxx	xxx	xxxx	xxxx	xxxx	xxx	xxx	xxx	xxx
	20	xxx	xxx	xxxx	xxx	xx	xxx	xxx	xxxx	xx
2	1	xx	xxx	xxx	xxx	xxx	xxx	xxxx	xxxx	xxx
	5	xxx	xxx	xxx	xxx	xx	xx	xxx	xxxx	xx
	10	xxx	xxx	xxx	xxxx	xxx	xxx	xxxx	xxx	xxx
	20	xx	xxxx	xxx	xxx	(2)	xxx	xxx	xxxx	xxx
3	1	xxx	xxx	xxxx	xxx	xxxx	xxxx	xxx	xxxx	xxx
	5	xxx	xxx	xxxx	?	xx	xxxx	xxx	xx	xxx
	10	xxx	xxx	xx	xxx	xxx	xxxx	xxx	xxx	—
	20	xx	xxxx	xxxx	xxx	xxx	xxx	xxx	xxxx	xxx
4	1	xxx	xxx	xxxx	xxx	xxx	xxx	xxx	xxx	xxx
	5	xxx	xxx	xxx	xxx	xxxx	xx	xxx	xxx	xx
	10	xxx	xxx	xxx	xxx	xxx	xxxx	xxx	xxx	xx
	20	xxx	xxx	(1)	xxx	xxxx	xxx	xxx	xxx	xxx
5	1	xx	xxx	xxx	xxx	xxx	xx	xxx	—	xx
	5	xxx	xxx	xx	xxxx	xx	xx	xx	xx	xx
	10	xxxx	xx	xxx	xxx	xxx	xx	xx	—	xx
	20	xxxx	xxx	xxx	xxxx	xxx	xx	xx	xxxx	xx
6	1	xx	xxxx	xx	xxx	xxx	xxx	xxx	xxx	xx
	5	xxx	xxx	xxx	xxxx	xxx	xxxx	xxx	xx	xxx
	10	xxx	xxxx	xxxx	xxx	xxx	xx	xx	xxx	xx
	20	xxx	xxx	xxx	xxxx	xxx	xxx	xxx	xxx	xxx
7	1	xx	xxx	xxx	xxxx	xxx	xxx	xxx	xxx	xxx
	5	xxx	xxx	xxx	xxx	xx	xxxx	xxx	x	xx
	10	xx	xxx	xxx	xxxx	xxx	xxxx	xx	xxxx	xx
	20	xx	xxx	xxx	xxx	xxx	xxx	xxx	xxx	xxx

(1) Died 4-28-36, lymphoid leukemia.
(2) Died 7-29-36, picked at vent by cage mate.
xxxx, xxx, xx, and x indicate excellent, good, medium, and poor "take," respectively, and — indicates no readable "take."

contact with other chickens or infectious material. Hence, these chickens were entirely suceptible. It was customary to use all of the chickens from one lot, until it was exhausted. Consequently, the age varied from six weeks to about four months, but in each titration the birds were of the same age. Chickens were banded and placed two to a cage for the titration.

The vaccine was applied by means of a swab and daily readings were made, which sometimes extended from the second to the tenth day. The majority of birds began to show a reaction at the end of 48 hours. The reaction increased in intensity, reaching a maximum usually on the fifth day. The sixth day, readings were noticeably less severe and sometimes by the eighth day all inflammation had disappeared. Although it is never necessary to test for immunity if a definite "take" has been produced, nevertheless the birds in the first four titrations of lots 1 to 7 were tested for immunity on the 9th, 13th, 12th, and 10th day, respectively, after vaccination by an intratracheal inoculation of 0.2 cc. of a virus suspension containing 20 mg. of dried virus per cc. In the remaining titrations questionable birds were inoculated, or their outcome indicated whether they were immune or not. To date, nine titrations have been made on lots 1 to 7, covering a period up to 421 days, or approximately 14 months. Since the fifth-day reading is the most significant, table II has been compiled on the basis of this reading.

An examination of table II shows that there was not always consistency between the concentration of virus and the severity of the local reaction, nor was there consistency with respect to the age of the virus. This may have been due in part to the age of the birds used. The bird which received a virus suspension of 5 mg. per cc. in the fourth titration and which never showed a definite "take" was nevertheless immunized. The two birds in the eighth titration which never showed the slightest suggestion of a "take" were shown to be immune to intratracheal inoculation. Moreover, before the immunity test was made blood serum was collected and this neutralized a virus suspension as determined by egg inoculation. The one negative reading in the ninth titration was definitely significant, for this bird contracted tracheitis from its cage mate. Lot 5 appears to have been the only one to suffer any loss of potency over the period of 14 months, and even this would still be a suitable vaccine. There appears to be no logical explanation for this decrease in potency.

Thus, of the 252 birds entered, 250 were alive for a fifth-day reading, and, of these, the reading was questionable in one and negative in three. And, only one of these four birds was a significant negative.

The results of the only two titrations made on lots 8 and 9 are given in table III.

TABLE III—Results of two titrations on lots 8 and 9.

Age of Virus (Days)		8	115
Titrations		1	2
Lot	Mg. Virus per cc.	12-29-36	4-15-37
8	5.0	xxxx	xx
	2.5	xx	xx
	1.25	xx	?
	.625	xx	xx
9	5.0	xxxx	xxx
	2.5	xxx	xx
	1.25	xxx	—
	.625	xxx	xx

The bird which gave the questionable reading later took the disease and the negative bird also took the disease and died on April 27. Here again there is inconsistency with respect to the concentration of virus used.

On the whole, the laboratory tests indicate that the immunizing power of virus propagated in eggs through 25 generations does not show any appreciable change. Furthermore, the tests on tenth generation virus show that when properly dried, the virus maintains its potency for more than a year, as determined by the method of inoculation used in routine immunization.

Field Trials

Seven commercial flocks containing over 5,000 birds were vaccinated with virus that was 128 to 460 days old. Here the virus

TABLE IV—Results of field trials with egg virus.

DATE	VACCINE LOT	AGE OF VACCINE (DAYS)	NUMBER VACCINATED	RESULTS
6-26-36	Pooled	128	300	Read about 50 on 4th and 5th day and 100% showed "takes"
6-29-36	4 and 7	131	500	About 10% read on 4th day and 100% showed "takes"
7- 2-36	5 and 6	134	1900	125 read at random out of 7 houses with one possible negative
7- 3-36	5	135	320	Two possible negatives
7-25-36	Pooled	157	1500	Two birds escaped vaccination; only 12 negatives in whole flock
7-28-36	Pooled	160	762	Percentage of "takes" not known but no losses from tracheitis occurred
5-24-37	5	460	134	All read on 5th day and 130 showed good "takes"

was used at the rate of 20 mg. per cc. of diluent. The results are set forth in table IV.

Unfortunately, every bird could not be read in five flocks, but in the sample readings made it is evident that a high percentage of "takes" was obtained. The flocks vaccinated on July 2 and 3 were on the college farm and we know definitely that no losses attributable to vaccination followed. Moreover, we have had no report of losses in any of these flocks.

CONCLUSIONS

Tenth generation egg virus dried and held *in vacuo* under refrigeration maintained its potency and immunizing power in laboratory experiments over a period of 421 days, even when used at the rate of 1 mg. per cc. of diluent. This virus also gave equally good results in field trials when used at the rate of 20 mg. per cc.

Twenty-fifth generation virus retained its immunizing power, but in the experiments the smaller doses employed gave inconsistent results.

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Wrens Hatch Family in Pocket of Overalls

In Bigsville, Ill., wrens hatched a family of five in a pocket of Postmaster Red Dixon's overalls and then vacated the garment. The overalls were hanging on a clothesline when the wrens started to build their nest and Dixon refused to disturb them.

The Metropolitan Life Insurance Company predicts that American women will grow taller. The women of today average an inch taller than their mothers.

Studies on Certain Filtrable Viruses. II. Immunization Against Fowl Pox with Fowl- and Pigeon-Pox Viruses Cultivated in Vivo and in Vitro*

By C. A. BRANDLY,† D.V.M., and G. L. DUNLAP,‡ D.V.M.

ALTHOUGH significant progress has been made in the prophylaxis of fowl pox during the past 15 years, consideration of work on the problem emphasizes certain inadequacies and shortcomings of the methods of immunization.

The value of unmodified, virulent, fowl-pox virus for vaccination is minimized by the potential hazards of impaired development and production as well as intercurrent disease, which tend to magnify undesirable systemic reactions. On the other hand, vaccination with pigeon-pox virus provokes in chickens only a modified pox reaction without serious constitutional disturbances, but the degree and duration of immunity are of such a low order as to limit greatly the value of the procedure. To date, attempts to attenuate fowl-pox virus by chemical or physical means have been unsuccessful, while the efforts to secure a desirably modified fowl or pigeon virus through biological modification (by serial passage in, or adaptation to, heterologous hosts) do not seem to have achieved the desideratum, namely, a virus vaccine with antigenic value equal to the unmodified fowl virus but with virulence so modified as to eliminate the constitutional disturbances commonly associated with unmodified fowl-virus vaccination.

That bacterial and/or virus contamination of skin-lesion pox virus employed for vaccination may be more or less dangerous is generally recognized. Methods which eliminate bacteria and at least greatly reduce the possibility of contamination with other viruses must therefore not be disregarded in the preparation and use of the virus vaccine. In this connection, the obvious advantages of egg-propagated or tissue-cultured virus as a source of vaccine are suggested. Furthermore, the possibility that cultivation of the viruses in pure culture under certain conditions may yield a desirably modified agent—one representing a fixed, innocuous and antigenically efficient variant—can not be overlooked.

Previously, one of the writers (Brandly, 1935) reported the results from a limited number of comparative tests using skin-lesion and egg-propagated fowl-pox virus for immunization. Since then, additional studies have been initiated, primarily with the goal of improving the safety of immunization procedures. The work described and the results here reported deal with the phases of the problem referred to and include work done largely in the laboratory.

MATERIALS AND METHODS

Sources of Pox Viruses Used.—Three strains of fowl-pox virus and one strain of pigeon-pox virus were employed. The pigeon strain was supplied in the form of skin-lesion scabs through courtesy of Dr. S. E. Eriksen and was believed to represent a strain originating in England. Beginning in March, 1937, it was carried through 15 chicken-egg passages.

Two of the fowl strains (1 and 2) were obtained from widely separated and apparently unrelated field outbreaks of fowl pox in chickens. Strain 1 was first propagated upon chicken eggs in March, 1935, and to date has been passaged at irregular intervals through 68 series of developing eggs, including one duck, one guinea fowl, and

*Funds used in the purchase of the eggs inoculated in the experiments at the University of Illinois were supplied by the Smith Incubator Company, Cleveland, Ohio.

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two turkey-egg passages. This strain was also maintained without egg passage by occasional propagation upon the skin of chickens, to provide a source of virus for determining possible alterations or modifications in character resulting from the egg passage.

Strain 2 has been passaged through 14 series of developing chicken eggs since April, 1937.

Fowl-pox virus strain 3 was secured in the form of commercial fowl-pox vaccine purchased on the open market and has been carried through 26 egg passages since July, 1936.

In order to free the skin-lesion pox virus of contaminating bacteria, susceptible birds were infected by skin puncture and the deeper portions of early skin pocks were excised aseptically, according to the method described by Woodruff and Goodpasture (1931). If bacterially sterile, this tissue was used for egg inoculation.

Technic of Fowl-and-Pigeon-Pox-Virus Propagation on Developing Eggs.—For cultivation of the pox viruses as well as for titrations, the method previously described (Brandly, 1935, 1936) was employed with few exceptions. In the latter case the technic described by Burnet (1934, 1936) was utilized. All of the eggs used for work included in this report were from a breeding flock of healthy, vigorous White Rock chickens. No cases of leukosis had occurred in this flock since it was established two years previous. Pullorum disease infection was eliminated by repeated testing.

Eggs showing actively developing, vigorous embryos were utilized for inoculation after twelve days of incubation. Following inoculation, the eggs were incubated at 37-38° C. with the large pole uppermost. The interval of incubation following inoculation varied from one to six days, depending upon the purpose of the experiment. The eggs were candled daily and dead embryos were removed promptly.

For brief storage the fresh virus tissues were held at a temperature of 0° to —4° C. with the exception of strain I, which was stored at 10-15° C. during a period of six months in 1936. Virus tissues for indefinite storage were dried at 0° to —4° C. in a vacuum of 370 to 410 mm. of mercury over a desiccating agent and then sealed in tubes and stored at the same temperature.

Tissue-Culture Propagation of Pigeon- and Fowl-Pox Viruses.—The medium of Li and Rivers (1930), as well as several modifications, were employed at the outset, but a minced chick-embryo-Tyrode solution medium, essentially the same as that described and used for vaccinia virus cultivation by Gallardo and Sanz (1937), gave the most satisfactory results in our hands. Briefly, the procedure is as follows: A living chick embryo 12 days of age is removed from the egg aseptically and minced finely in a watch glass encased in a Petri dish. Approximately 8 cc. of freshly filtered Tyrode solution is added. Of this mixture, 0.25 cc. is added to 4.25 cc. of Tyrode solution, previously placed in 50-cc. Erlenmeyer or Rivers collar flasks, which are fitted with gauze-covered cotton plugs and with rubber caps. To initiate the *in vitro* cultures, 0.3- to 0.5-cc. quantities of 1:100 suspension of bacteria-free pox-infected chorioallantoic membranes were transferred to each flask. For seeding flasks of fresh medium 1-cc. quantities of the culture were transferred directly. For larger flasks (125 cc. Erlenmeyer and 250 cc. Rivers) corresponding amounts of medium and inoculum were used.

Following inoculation, the flasks were held at 10-15° C. for 24 hours and subsequently transferred to the 37-38° C. incubator. After incubation for periods of three to five days, the flasks were removed to the 10-12° C. ice chest and, if used for inoculation of tissue cultures, eggs or chickens, this procedure was carried out within the week following the termination of the incubation period. For immunization experiments, the entire culture, fluid and minced tissue were utilized. In preparing the tissue-cultured virus for animal inoculation and egg inoculation, the fluid was removed and the minced tissue ground in small glazed mortars, after which it was again mixed with the fluid portion.

Some flasks in certain series failed to show detectable multiplication of the virus and, in one instance, all of the flasks of

TABLE I—Comparative titration of pox viruses.

Exp.	Passage Upon			End-Point Dilution Infective for	
	Virus	Eggs	Tissue Cultures	Eggs	Chickens and Pigeons
1	Fowl pox I	62	—	10^6	—
1		4	—	10^4	—
2	Fowl pox I	63	—	10^4	—
2		5	—	10^5	—
3	Fowl pox I	64	—	10^4	—
3	Fowl pox II	4	—	3×10^4	—
4	Fowl pox I	60	—	10^5	—
4		—	3	10^3	—
5	Pigeon	11	—	10^5	—
5	Pigeon	3	—	10^5	—
6	Pigeon	12	—	10^5	10^5 (pigeons) * 10^3 (chickens)†
6	Pigeon	—	4	10^2	10^2 (pigeons)*
6	Pigeon skin	—	—	—	10^3 (pigeons)*
6	Fowl pox II	11	—	10^6	10^5 (chickens)
6	Fowl pox I	66	—	10^6	10^4 (chickens)
6	Fowl pox I	—	6	10^3	10^4 (chickens)
6	Fowl pox I comb	—	—	—	10^2 (chickens)
7	Fowl pox I	58 + 61	—	10^5	10^3 (chickens)
7	Fowl pox I (skin scabs)	—	—	—	1.5×10^2 (chickens)

*Inoculation upon scarified plucked breast. All other chickens inoculated by stick method.

†Feather-follicle inoculation.

the fifth transfer series were inactive on testing. However, by careful technic and frequent determination of virus concentrations upon developing eggs, continuous cultivation of an active virus was not found especially difficult. Nevertheless, the method of chorioallantoic propagation of the pox viruses was much more satisfactory from the standpoint of ease of maintaining the active agent in pure culture.

Observations on Fowl and Pigeon Viruses Before and After Tissue Culture and Developing Egg Passage

Several experiments were conducted with susceptible chickens as well as with developing eggs to determine whether cultivation in eggs or tissue culture by the procedures outlined had affected significant modifications of certain properties of the viruses. It was assumed that within certain limits alterations or modifications of virulence or infectivity would be expressed by differences in the virus concentrations of infected tissues and in the extent and nature of the lesions produced. Consequently, comparisons were carried out with egg membranes infected by the same and different strains of virus which had undergone few and numerous egg transfers. Homologous virus from pigeon- and chicken-skin lesions was also tested with egg-propagated and tissue-cultured virus.

Comparative Titrations on Eggs and on Birds.—For preparation in testing, virus-infected tissues were ground finely in small mortars and suspended in nine parts by weight of broth. Upon centrifugation at 2,500 r.p.m. for five minutes in a horizontal centrifuge, the supernatant fluid was removed for suitable dilution in broth.

Four or more eggs were utilized for inoculation with each dilution in the titrations on eggs. All eggs, including those with embryos succumbing prior to the sixth day, were opened and examined at the sixth day following inoculation. Determination of titre was considered valid only when at least two eggs of each end-point series and of the series receiving the next greater concentration of virus showed typical macroscopic lesions of the chorioallantoic membrane characteristic of pox.

In titrations on chickens, birds 6 to 12 weeks of age were employed and inoculations of the various dilutions on four or more well separated areas of unfeathered skin on each side of the body were accom-

plished by a suitable number of skin punctures. Usually two birds, but occasionally only one, were used for comparisons, dilutions of one virus being applied to the left side of the body, the other to the right. In titrations of pigeon virus on pigeons, usually only one dilution of each virus was tested on a single bird. The dilutions were applied to the plucked, scarified breast of each side. For measurements of pigeon virus on chickens the skin of the thigh was pricked and scarified in a manner similar to that employed for pigeons. Observations were made during the period from three to fifteen days following exposure.

The results of the various titrations are summarized in table I.

In titrations of this nature considerable quantitative variations of the virus factor as well as certain individual differences in resistance of embryo tissue and of birds are to be expected. However, it is believed that hundred-fold or greater but not ten-fold differences of infectivity, as obtained in certain individual titrations, may be of possible significance. Nevertheless, where several titrations, as in the case with few and many egg-passaged strains of virus, failed consistently to manifest titre differences of hundred-fold or greater, valid distinctions in infective potency could not be set up.

The titrations with infected chorioallantoic tissues apparently failed to reveal any significant differences in the virus content or infectivity of egg membranes representing few as compared to numerous egg passages. The pigeon-virus comparisons, although representing a much narrower range of egg passages, also failed to show any detectable differences.

From the limited comparisons in experiments 6 and 7, table II, it appears that the chorioallantoic membrane of 12-day chicken eggs is more susceptible to fowl- and pigeon-pox virus than is the skin of chickens. In the single trial with pigeon virus (experiment 6) differences of similar order in the susceptibility of the skin of pigeons and the membranes of chicken eggs did not appear. That this may be accounted for in part by the natural host tissue affinity of the pigeon virus is suggested in this experiment by the apparent greater susceptibility of the pigeon over the chicken skin. The desirability of including developing pigeon eggs is pointed out in further study of this point.

There would not appear to be any significant differences in the potency of few and many egg-passaged virus of strain II. Strain II, although carried only through limited egg passages, would seem to possess a potency similar to strain I.

Regarding concentrations of virus in chorioallantoic membranes, tissue cultures, and skin lesions, the membranes were found to be definitely and quite regularly richer in virus. The titres of the tissue-cultured fowl-pox virus strain I were from ten to one thousand-fold less than with the egg propagated, but the virus concentration of the tissue cultures generally exceeded that of skin-lesion scabs. The possibility that simple dilution would account for the differences in tissue-cultured and egg-type virus is excluded by the similar potency of the third and sixth tissue-culture passages. Furthermore, the original inoculum represented a dilution of at least 4×10^5 in the sixth tissue-culture passages.

Observations on the Nature and Extent of Lesions.—The lesions of the chorioallantoic membrane were observed to determine if changes in nature or extent of primary, as well as secondary, lesions had occurred as a result of egg- and tissue-culture passages.

Marked macroscopic changes or variations were not detected in the nature of the lesions produced in the membranes by the few or many passaged strain I fowl-pox virus. However, there seemed to be a somewhat greater tendency toward necrosis and metastasis with long-continued passage and apparently because of more extensive involvement of the chorioallantois with consequent greater interference of its respiratory function, embryo mortality occurred as an early and regular result in the later egg passage of this virus. Strain II fowl-pox virus of a limited number of egg passages produced gross and microscopic lesions identical with those of early-passage strain I

virus. The results were similar with the strain I tissue-cultured virus employed. However, the strain I virus had been passaged through more than 50 series of eggs prior to cultivation on tissues *in vitro*.

On gross observations the well advanced pigeon-virus lesions of the chorioallantois showed evidence of greater cellular proliferation and less necrosis than the lesions of fowl pox. The metastatic tendency was very slight and did not appear to be greater after 15 passages of the pigeon virus than in the first few passages. Some focal lesions produced by the pigeon virus were 15-20 mm. in diameter, flat or slightly convex on the entodermal surface, while the ectodermal aspect represented almost a true hemisphere.

Microscopic examinations of a limited number of the chorioallantoic membranes infected with fowl- and pigeon-pox viruses verified this gross observation. Both types of viruses produced retrograde changes in all three germ layers. Fowl-pox virus produced more severe changes in the ectoderm and entoderm, while the pigeon virus evoked a more severe reaction in the mesoderm. In making this observation it is recognized that inoculations of the virus may have represented introduction within the membranes as well as upon the ectoderm. Necrosis of the limiting epithelial layers was apparently associated with the development of cytoplasmic inclusions. In these comparisons the fowl-pox strains of virus produced larger and more easily demonstrated inclusions in the ectoderm and entoderm than did pigeon-pox virus. Involvement of the entoderm in pigeon-virus lesions was slight. The observations on a considerable number of egg membranes suggest that if numerous egg passages had produced any significant change as far as the fowl-pox infective process within the egg membranes was concerned, these alterations were expressed in slightly more rapid development of the pocks and a greater tendency toward metastasis.

In previous work with the strain I of fowl-pox virus, significant modifications in chicken-skin lesions were not observed as a result of eight egg passages (Brandly, 1936). With further egg passage, additional comparisons were made in conjunction with vaccination experiments, and in several inoculations of chickens expressly for the purpose of studying the development and recession of the pocks.

The results in a small number of experimental birds and in several field flocks did not reveal any differences in severity and extent of the local response to inoculation which could not be attributed to the different concentrations of the virus in certain lots of egg-propagated, tissue-cultured and skin-lesion virus. The more rapid development of lesions obtained with various egg and tissue-cultured virus as compared to other suspensions could likewise be explained on the basis of virus concentration of the inoculum.

COMPARISONS OF THE EFFECTS OF IN VITRO AND IN VIVO CULTURED VIRUSES ADMINISTERED TO CHICKENS

In view of the results reported by Doyle and Minett (1927), Basset (1924, 1928, 1935), and others on the effect of inoculation of skin-lesion viruses by various routes, it was deemed desirable to determine the effects of intramuscular and dermal inoculation of the various types of fowl and pigeon-pox viruses. It was expected that inoculation of various quantities of the different types of virus-carrying material into experimental chickens 10 to 12 weeks old would allow comparisons and indicate with some accuracy the dosage which, in the case of the intramuscular route, would be reasonably safe yet effective for use in immunization experiments on a larger scale. Undiluted tissue-cultured virus, 1 per cent suspensions of skin-lesion virus and 1 per cent suspensions of egg-membrane virus constituted the inocula. Quantities of 0.25 and 0.5 cc. of the various virus suspensions were injected intramuscularly into duplicate pairs of birds. For follicle inoculation with fowl virus, four to six open follicles, and with pigeon virus, eight to twelve open follicles were employed, while two "sticks" constituted the puncture method of dermal exposure.

The results failed to show any significant

TABLE II—Summary of results with the use of egg-propagated, tissue-cultured and skin fowl-pox virus in vaccination.

GROUP	NO. BIRDS	AGE	VIRUS	ROUTE	PERCENTAGE SATISFACTORY "TAKES"	NO. SHOWING SECONDARY POX	MORTALITY		IMMUNITY TESTS OF 10 BIRDS
							NO.	%	
1	40	5 wks.	SI egg 1:100	i. m.	——	3	20	50	Immune
2	40	5 wks.	SI egg 1:100	stick	100	0	22	55	Immune
3	40	5 wks.	SI egg 1:100	f. f.	100	0	14	35	Immune
4	40	5 wks.	SI t. c. undil.	i. m.	——	2	24	60	Immune
5	40	5 wks.	SI t. c. undil.	stick	95	3	10	25	Immune
6	40	5 wks.	SII egg 1:100	i. m.	——	4	12	30	Immune
7	40	5 wks.	SII egg 1:100	stick	100	2	7	17.5	Immune
8	40	5 wks.	SII egg 1:100	f. f.	100	1	4	10	Immune
9	40	5 wks.	SI skin 1:100	stick	90	4	12	30	Immune
10	40	5 wks.	SI t. c. 1:10	i. m.	——	10	13	32.5	Immune
11	40	5 wks.	SI t. c. 1:10	stick	94.7	3	5	12.5	Immune
12	40	5 wks.	SI t. c. 1:10	f. f.	100	0	16	40	Immune
13	40	5 wks.	— Controls	—	——	—	5	12.5	Not immune

i. m. = Intramuscular.
Stick = 2 sticks in skin of breast.
f. f. = 4-6 feather follicles.
t. c. = Tissue culture.

difference in local lesions and weights of the birds after vaccination with the three types of virus by the stick and by the feather-follicle method. The weight gains in both groups were approximately one-half as great as those of the uninoculated controls. Good "takes" developed in the vaccinated groups.

With intramuscular inoculation the groups given the egg-membrane virus and the smaller dosage of tissue-cultured fowl virus showed weight gains similar to those treated cutaneously. The groups treated with the skin virus and larger dosage of tissue-culture virus failed to gain weight during the period of three weeks following vaccination. Tests for immunity to heavy cutaneous exposure after six weeks showed all of the surviving birds of the control group to be susceptible, while the treated groups were apparently completely immune to fowl pox.

With tissue-cultured and egg-propagated pigeon virus the "takes" obtained by feather-follicle inoculation showed considerably more swelling after one and two weeks than did those from skin-lesion virus. Moderate scabs of the follicles were produced, those from the egg and tissue-culture virus being slightly larger and thicker at the second week. All birds treated with tissue-cultured and skin-lesion virus showed slight losses in weight when weighed at the end of the first and second week. In contrast, the egg-membrane-virus treated and control birds showed slight weight gains.

In spite of individual intramuscular dosages as high as 10 cc. of the tissue-cultured or egg-propagated pigeon virus, the weight gains in ten birds treated in this manner were affected only slightly, if at all. In contrast, the birds receiving 0.25 and 1 cc. of the skin-lesion virus all showed moderate to marked weight losses at the end of one and two weeks after injection.

Tests for immunity to fowl pox at three weeks following vaccination gave somewhat irregular results. A small percentage of birds, including individuals treated intramuscularly and by feather-follicle inoculation with skin virus, were as susceptible or only slightly less so than the controls. Two birds which had received 10-cc. quantities of egg-grown pigeon virus intramuscularly showed a slight but definite lesion to fowl-pox exposure.

IMMUNIZATION OF LARGER GROUPS OF EXPERIMENTAL CHICKENS

(a) Fowl-Pox Virus Inoculation

With the results of preliminary experiments available, larger groups of chickens 5 to 6 weeks of age, composed largely of White Leghorns, were vaccinated. Each lot was divided into groups corresponding closely with regard to breed, proportion of

sexes and total weights. The strain I egg virus represented the 66th egg passage, the strain I tissue-cultured virus the sixth passage, and the strain II virus, the eleventh egg passage. The strain I skin virus consisted of fresh skin-lesion scabs harvested three weeks before use. Titrations of these lots, previously made, are recorded in table I.

Interpretations of puncture and feather-follicle "takes" were on the arbitrary basis of reactions classed as excellent, good, fair, slight or doubtful, and negative. Only fair, good, and excellent "takes" were considered satisfactory. The weights of the surviving birds were taken and tests were made for immunity (with strain II pox-infected egg membrane) at six weeks following vaccination. The mortality records embraced the six-week post vaccination period. Autopsy examinations did not reveal any factors of mortality other than coccidiosis and fowl pox.

The birds in groups of 80 and 120 were housed in unheated, outside 12′ x 12′ houses during late spring. A period of cool, rainy weather, together with an attack of cecal coccidiosis in all of the houses, including the controls, contributed to mortality of serious proportions in spite of daily cleaning subsequent to the appearance of the outbreak. The major losses occurred between the second and fifth week after vaccination. It is recognized that the effects of coccidiosis and environmental factors may render questionable certain determinations, *e. g.*, those on weight gains and mortality. Nevertheless, we believe that the data as a whole provide some definite information.

An outline of the procedure, together with the results as recorded according to the percentage of "takes," mortality, number showing secondary or inoculation pox, and immunity tests are summarized in table II.

Although the differences were slight, it appears that the egg-propagated fowl viruses, both I and II, were more efficient than the tissue-cultured and skin virus, strain I, in producing "takes" by the stick method. Distinctions between various degrees of a positive reaction to stick or puncture are, of course, easier to make than in the case of the feather-follicle reactions. This could account in part for the interpreted greater efficiency of the latter procedure in producing "takes."

On the basis of secondary pox-lesion production in these groups, intramuscular inoculation would seem to be much less desirable than the other methods, regardless of the type of fowl-pox virus used. The fact can not be overlooked that the quantities of virus which were used represented a dosage at least several thousand-fold greater than necessary to produce skin infection, although these amounts were not injurious to the older stronger birds in the preliminary experiment. Nevertheless, in view of Basset's reports (1924, 1928, 1935), the effects of smaller quantities of bacteria-free virus introduced intramuscularly should be given further study.

As already pointed out, attempts at correlation of mortality figures with methods of inoculations and types of virus used do not seem to be warranted. The hazard of intercurrent disease and other devitalizing factors recognized by all workers who have dealt with this problem are well emphasized here. The results of weight-gain determinations which were made were also disregarded for this reason and are not included in the table. It is, however, considered significant that ten birds from all groups, 1 to 12 inclusive, were immune to stick-method exposure with egg-propagated fowl-pox virus when tested at the end of the sixth week following vaccination. The test birds from groups 5, 9, and 11 included one or more individuals not showing a satisfactory "take" from stick vaccination. The controls (lot 13) were susceptible, as indicated by fair to good "takes" one week after inoculation with the test virus.

(b) Pigeon-Pox-Virus Inoculation

Chickens from the same source, age, and breed which were used in part (a) constituted the first lot used for pigeon-pox inoculation. The second lot (groups 5 to 8 inclusive in table III) were composed of about equal numbers of Rhode Island Reds

and Barred Rocks. The entire procedure was identical with that described for the fowl-pox series, except that in stick vaccination, four punctures were made instead of two. In the feather-follicle inoculation, six to eight open follicles were treated. Groups 5, 6, and 7 were vaccinated one week later than the first three groups. The procedure and results are outlined in table III.

TABLE III—Summary of results from the use of egg-propagated pigeon-pox virus.

GROUP	NO. BIRDS	AGE	VIRUS	ROUTE	PERCENTAGE SATISFACTORY "TAKES"	MORTALITY		IMMUNITY TESTS (7 WKS.) OF 10 BIRDS FROM EACH GROUP
						No.	%	
1	40	5 wks.	Egg 10%	i. m.[x]	——	4	10	4 S, 6 N
2	40	5 wks.	Egg 10%	Stick	0	9	22.5	4 F, 5 S, 1 N
3	40	5 wks.	Egg 10%	f. f.	95	8	20	4 S, 6 N
4	40	5 wks.	Controls	—	——	4	10	6 G, 4 F
5	40	6 wks.	Egg 5%	i. m.[x]	——	10	25	3 G, 3 F, 4 S
6	40	6 wks.	Egg 5%	Stick	22.5	11	27.5	3 G, 5 F, 4 S
7	40	6 wks.	Egg 5%	f. f.	95	9	22.5	2 G, 1 F, 6 S
8	40	6 wks.	Controls	—	——	5	12.5	2 E, 4 G, 4 F

x = 0.25 cc. injected into muscles of breast of each bird.
f. f. = Feather follicle (six to eight follicles). i. m. = Intramuscular.
G = Good; F = Fair; S = Slight take; N = Negative reaction to fowl-pox inoculation; E = Excellent.

Secondary pocks at or near the site of inoculation or upon the integument of other portions of the body were not observed in any of the groups treated with pigeon virus. Failure to obtain satisfactory "takes" with stick or skin-puncture methods, even with relatively concentrated suspensions of pigeon-pox virus, was perhaps to be expected in view of similar results previously observed by various investigators. Any apparent differences between groups 2 and 6 inoculated by the stick method may be considered negligible. In groups 1 and 5, tincture of iodine was applied to the puncture immediately after intramuscular inoculation to reduce the possibility of skin "takes."

Since inoculation of chickens with pigeon-pox virus generally does not effect a significant systemic reaction, the losses in the various groups were attributed largely to the coccidial infestation which occurred subsequent to vaccination. No differences of any consequence, with the possible exception of group 2, are observed, as far as the influence of virus concentrations or routes of inoculation are concerned.

The identical results of the immunity tests in groups 1 and 3 suggest the presence at seven weeks of a similar definite but not absolute degree of immunity to artificial exposure to strain II of fowl pox. The interpretations of the "takes" in birds first treated by the stick method suggest only a slight or doubtful degree of resistance.

The results in lots 5, 6, and 7 would indicate the production of only a partial immunity to subsequent artificial exposure to fowl pox.

DISCUSSION

The findings here recorded would seem to emphasize several points of superiority of the egg-propagated fowl-pox virus over the skin-lesion virus generally employed at present for immunization against fowl pox. The pox-infected chorioallantoic membranes were invariably richer in active virus than the skin-lesion tissue of a similar age and method of handling. Strain I fowl-pox virus was cultivated through 68 successive series of eggs without any apparent reduction in its pathogenicity or virulence for developing eggs or chickens.

Although fowl and pigeon-pox virus may be cultivated in tissue cultures *in vitro* in concentrations suitable for immunization procedures, this method was not as satis-

factory in our experience as propagation in developing chicken eggs.

Different workers (Harrison and Streit, 1903; Beach *et al*, 1915; Crofton, 1924; Basset, 1924; Pyle, 1926; and others) have reported the presence of various bacteria, including *Pseudomonas aeruginosa, (Bacillus pyocyaneous), Staphylococcus aureus* and *albus, streptococci, Escherichia coli, B. cacosmus,* and *Pasteurella avicida*-like organisms in fowl-pox skin lesions or in vaccine prepared from the skin lesions. The possibility of infection with the bacteria associated with fowl-pox skin-lesion virus vaccine when the agent is applied cutaneously has been suggested, but not established to be of serious consequence. However, Basset (1924) considered voluminous and persistent lesions during the course of avian pox to be a consequence of secondary microbes, while Pyle (1926) concluded that the secondary staphylococci and *P. aeruginosa* assisted the virus in causing a shorter period of incubation and a more pronounced form of fowl pox.

Rasch (1930) reported *B. enteritidis,* Breslau type, associated with pigeon-pox vaccine and the death of pigeons treated on the skin of the breast with this vaccine. The organism was recovered from the latter birds in pure culture. Stubbs (1935) found that the virus of leukosis, present in large quantities in the blood of some affected chickens, may resist drying and temperature conditions similar to those to which skin-lesion virus is subjected prior to use for vaccination. However, the hazard of direct transfer of certain forms of leukosis within the flock by pox-vaccination procedures, as demonstrated by Johnson (1937), will require additional precautions.

In this connection, the use of a metal inoculating instrument, such as that suggested by Fleischhauer (1932), may be invaluable to overcome direct transfer of leukosis and other infections during the process of vaccination. Fleischhauer suggested sterilization of the instrument by the use of a spirit burner after treating each bird to avoid transfer of pox infection already present.

If it is granted that the potential dangers of infection or injury by the bacteria associated with skin-lesion virus vaccine may not be particularly significant in the present methods of cutaneous vaccination, it is nevertheless apparent that the egg-propagated and tissue-culture viruses as a source of bacteria-free virus vaccine represent a distinct and desirable refinement. Our own observations suggest that with proper precautions in the selection and management of the breeding stock, possible contamination *via* the egg with other viruses, such as leukosis, may very probably be excluded entirely. Certainly, the hazard would seem to be less than in the use of chickens in producing the skin-lesion virus vaccine.

The facility with which pigeon-pox virus may be cultivated upon the extraembryonic membranes of developing chicken eggs should allow additional information to be obtained upon the practical merits and limitations of this agent in the fowl-pox control program. The high virus concentrations of the infected chorioallantoic membrane apparently render this virus material more satisfactory than the pigeon-skin-lesion virus for immunization studies in chickens.

Our results with limited trials of intramuscular introduction as compared to the dermal routes (stick and feather follicle) did not concur with Basset's findings that fowl virus could be employed to better advantage by the former method. However, generalized pox lesions and severe secondary reactions after intramuscular inoculation experiments could be attributed in part to excessive dosages and intercurrent disease, since in a limited number of tests with 10-week-old chickens of normal vigor, the intramuscular inoculation of pure culture fowl-pox virus in quantities a thousand-fold greater than the inoculum introduced by dermal vaccination did not induce systemic reactions any more severe than those resulting from dermal vaccination. Massive doses of pure culture pigeon-pox virus introduced intramuscularly in chickens failed to induce a significant systemic response and would suggest that inoculation pox and severe systemic reaction following pigeon-pox-virus vaccination, as reported by Glover

(1931), were probably due to intercurrent fowl pox infection.

SUMMARY

1. In these experiments bacteria-free egg-propagated and tissue-cultured as well as skin-lesion (bacteria-carrying) fowl and pigeon viruses were employed to determine their value for immunization of chickens against fowl pox. The former appear to possess several points of superiority.

2. The chorioallantoic membranes of 12-day eggs were found to be more susceptible to infection with fowl and pigeon-virus than was the skin of chickens 6 to 12 weeks old. Furthermore, the egg-propagated virus material (chorioallantoic membrane lesions) almost invariably was found to be richer in virus than the tissue-cultured and the skin-lesion virus material tested.

3. Large intramuscular doses of the bacteria-free forms of virus appeared to show less tendency to cause undesirable constitutional reactions in vigorous 8- to 12-week-old chickens than did the bacteria-contaminated skin-lesion virus material.

4. As compared to the lesions produced in the chorioallantoic membranes of 12-day chicken eggs by fowl-pox virus, the pigeon-virus lesions showed much greater cellular proliferation and much less tendency to necrosis and secondary metastatic foci.

5. Passage of one strain of fowl-pox virus through 68 series of developing eggs over a period of about two years did not appear to bring about any significant change in virulence for chickens or the chorioallantoic membrane of developing eggs.

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Tetanus Toxoid, the Antitetanic Product of Choice

Vaccination against tetanus with tetanus toxoid is the method of choice in man and animals. The protection engendered is permanent. It removes the danger of contracting that disease from open wounds or from inapparent tetanogenous foci. Tetanus toxoid may be given with other vaccines (typhoid, diphtheria, etc.) and is therefore a useful adjunct in group vaccinations.

The opportunity afforded by mass vaccination of horses of armies has furnished indisputable proof of its efficacy. Since 1927, the use of it on a large scale in the horses of the French army has made tetanus disappear from the army mounts. A single, nonfatal case occurred in 1937. (*G. Ramon. Tetanus Anatoxin and Prophylaxis in Man and in Domestic Animals. La Presse Medicale, xlvii, June 3, 1939. Abstract in the Journal of the American Medical Association.*)

National conferences on child welfare are held in Washington, under presidential auspices, at intervals of approximately ten years. The fourth conference of the series was formally opened by President Roosevelt at the White House the past April.

"Dos and Don'ts" for the Practicing Veterinarian

By W. C. GLENNEY, D.V.M.
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THE NUMEROUS small things that are a part of the daily routine seem to determine the success of a practitioner. These are the "dos and don'ts" which this paper intends to consider.

A good many difficult clients develop into fine clients by careful handling. Their faith in veterinary service may have been jarred and, whether justifiable or not, it is up to the present doctor to restore that faith. He owes it to the profession—not to mention the possibilities of a new client. With the progressive improvement in service and equipment, the public's faith has been strengthened, but another generation will have to pass on before clients cease to utter, "Did you know Dr. ——— years ago? Well, anyway, I'll never forget the time he strapped my dog to his barn and spayed her without any anesthetic." This is not cited to condemn the practitioners of bygone days. They did as well as they could with the facilities at hand. They had a reason to allow for such things, but with the present situation our "slips" should be in the minority. The ascending standards of veterinary medicine demand caution and accuracy in every detail of practice.

The matter of office hours is an important consideration, particularly in the case of the small animal practitioner. Recently, however, a large animal practitioner was bemoaning the fact that his clientele made his office an evening gossip center. This was remedied by closing at a scheduled hour and handling only emergencies after those hours. Where there is but a single country office, it is impossible to discuss freely with a client matters which should be confidential. As a solution, there should be a sub-office to handle clients that do not wish to confide before an audience. Once you have established definite hours, be certain to maintain them. Do not disappoint your clients. Once a client depends on you or an assistant to be available at certain hours, return the courtesy by having someone on the job. During rush seasons in the country, however, office hours are of necessity uncertain. They must be sacrificed in favor of work that can not be postponed.

During the recent encephalomyelitis vaccinations, horses were treated anywhere at any time. Often a heated horse received a potent vaccine that should have been administered to a cool animal and, thus, systemic or localized reactions occurred that could have been prevented.

When possible, hog vaccination should be done during the cooler parts of the day. Barring emergencies, major surgery should be done in the forenoon in order to allow time to observe the patient before night.

A significant factor in successful practice is personal appearance. Regardless of your status in life, cleanliness is expected, and particularly in the professional world. Neatness in dress is admirable and not necessarily expensive.

An untidy car and equipment carelessly strewn about have a definite connotation. To those who claim, "I am too busy to have my car washed or to clean my equipment," it might be pointed out that it is better to slacken the pace a bit than eventually to have no practice and plenty of time to clean up.

In our locality, the dentists and physicians prove to be good clients. However, we find it difficult to establish rates for them. As a general rule, they are broad-minded, and because our rates are so much less than the human rates, the matter is usually settled peacefully at the regular fees. They recognize good service and, particularly, if up-to-date equipment is used, they are willing to pay any reasonable rate.

(Continued on page 366)

EDITORIAL

The justification of any medical organization lies in the unselfishness of its ideals, it achievements come from its initiative and freedom from guild fundamentalism, and its permanence rests upon its service to the public.—Kanavel.

The Rabies Question

IN THE OPERATIONS of the veterinary service, the dog is not just another species of domestic animal. It is a sort of deified member of the household, the pride of its owner, and the consecrated toy of the nursery, not to mention its high place in the field of sport, and in definite utilitarian pursuits of peace and war. In these several estates, by common consent, the dog is held aloof of the forensic regulations governing the possession of other animals. Before the law, it is the sacrosanct animal that was first to yield to domestication and, certainly, the first to win a permanent place in the hearts of its masters. Free love and roaming are sacred rights and woe be to him who proposes reforms. That the "phobes" and the "philes" will lurch into verbal paroxysms when their respective ideas are challenged is proof that the dog is not just another domestic animal in veterinary prophylactic medicine.

In protecting live stock against the raids of threatening diseases, the veterinary service is necessarily uncompromising. All sentiment is apt to be set aside to accomplish its purpose. The owners yield for the public good. Only an adjustment in dollars and cents stands between the owner and the law. Not so with the dog, for here is a domestic animal protected by a psychological barrier that enters into the calculations and which can not be rudely overlooked in striving to arrive at the desired objective. That is to say, any attempt to control a disease of the dog that does not reckon with the age-old sentimentality associated therewith must of necessity interfere with the project, if not defeat it entirely in a democratic country like ours, where the will (sentiment) of the people rules.

Vaccination and other measures employed to conquer rabies, for example, have their proponents. Though truth based upon scientifically established fact should be the rule and guide of all concerned, exaggerated misinformation too often enters into the arguments of both sides. Powerful adjectives are invoked in defense of opposing ideas. On the one side is the determination to control disease in animals (the duty of the veterinary service) and on the other side is sentimentality surrounding the possession of "man's best friend." In this case, the veterinary service has a condition of mind as well as an ugly disease to handle.

That rabies is a neglected disease in the United States is an unassailable indictment of the veterinary service. The sentimentality reigning in dogdom appears to be the responsible factor, for, in the matter of enforcing the standard disease-control measures, this is a branch of animal production that is not as easy to discipline as the branches furnishing food, motor power and transportation. Hence, in order to bring rabies under control in this country to the same extent as in England and France, dogdom and the veterinary service will have to come to an understanding. Dogdom has the choice between controlling the disease or fighting an uphill battle against it. Veterinarians who have lost client after client through a visitation of this gruesome disease know that this fear is not fiction.

RABIES AND THE A.V.M.A.

The annals of veterinary medicine in recent times have been distinguished by some major disease-control project which naturally became the main topic of our successive annual conventions. An outstanding dis-

ease of live stock or some important phase of its nature or of its control stands out on the planned programs of the Association each year.

There have been periods when Texas fever, tuberculosis, glanders, foot-and-mouth disease, epizoötic abortion and mastitis were the feature subjects of our meetings. A glance through the programs of the past shows how alert our national association has been in collecting knowledge that can be used for the conservation of the nation's animal industry. Important as all of these still remain, they can be set apart as disease systematically and effectively handled as the Association goes on exploring new fields of the same genre and making new recommendations. The new field this year is rabies in dogs.

When this issue of the JOURNAL arrives, the Association will be dramatizing rabies control at Memphis. A special committee composed of connoisseurs of the disease will be inscribing its opinion on the records, and prominent figures in the realm of scientific investigation will be reading results of extensive investigation. Obviously, these reports and the extemporaneous discussions will make the 76th annual meeting of the Association a new point of departure in the control of rabies in animals.

Rabies affects many species of animals and man, but there is only one element to control, and that is the bite of the rabid dog. This accomplished, rabies should vanish from the country as it has from other countries where that element was abolished. It is therefore the duty of the American Veterinary Medical Association to keep the control of rabies at the top of its trestleboard until that utopian objective has been reached, just as it has been reached for the other animal plagues mentioned above.

The JOURNAL is wide open for arguments on all sides of this question based upon research, statistics and clinical data that are needed to set all fair-minded groups working for the desired end. The object should be to circumvent the broadcasting of unsupported information to the detriment of both the veterinary service and the canine branch of animal production.

With rabies running amok in the United States, dogdom hasn't a ghost of a chance to win universal popularity for dogs which, needless to tell the readers of this journal, is the outstanding aim of the veterinary profession in regard to this or any other species of domestic animal. The insinuation that veterinarians maneuver their actions to make money is "old stuff" that was never justified by the ends attained. In our drive through these insinuations we have had to push them aside and go on protecting public welfare.

Though the scientific principles involved in canine prophylactic medicine are precisely the same as those of other branches, the medico-legal and sentimental factors have to be considered in the work of making the diseases of dogs less noxious to man and other animals, because in livestock sanitary science and police, the dog has been a bird of another feather as far back as the era of prehistory. This fact was never more clearly proved than by the tirades against regulatory measures affecting dogs.

Register Your Ideas

THE EXECUTIVE office solicits correspondence from the members on all sorts of subjects, for it is through that intermediary that we shall be able to boil down the *status quo* to a useful end. In the words of Wendell Phillips, "It is only liquid currents of thought that move men and the world." Silence is golden only when one has nothing worth saying. And, how can we guess at what you are thinking about?

THE PROGRAMS carried out by scientific societies at their conventions lean toward open discussion rather than the reading of long manuscripts. In defense of this reform, convention-goers point out that to follow even the most coherent scientific paper is a mental strain, and that they are able to derive greater benefit from such articles by reading and studying them at leisure.

The Veterinary Profession*

THE VETERINARY profession is a standing army, oriented practically unnoticed, between failure and success in farming. Its members keep sweeping contagions of live stock under control and certify to the wholesomeness of most of the meat and milk consumed, besides furnishing practitioners to attend to the sick and injured.

The farmer of today, like the nation of all times, who compromises with the diseases of his animals does not prosper, while those who obey the doctrines of veterinary science thrive and remain self-contained. . . . Those of us who have watched the ups and downs of American farming through the years are disappointed that the farmers of our country have never been able to impress the people with the fact that the fate of our country lies in the successful production of domestic animals; and this only a well disciplined veterinary service can furnish. . . . The main objective of the veterinary profession is to make farming a profitable occupation through the production of healthier and better live stock, the medium through which so large a percentage of farm crops reach the consumer and which supplies most of our food, much of our clothing, all of our pets, and our animals of sport, pleasure and general utility. . . . Trained veterinarians know the leaks which make farmers work hard for small profits. They see the stealth by which apparent and inapparent disease robs the farmer of much of his income, and therefor they invoke the farmers' aid in building up a competent veterinary service. . . . The veterinarians through their national and state societies solicit the aid of the farmers' organizations and the leaders in the science and art of agriculture, knowing that animal diseases are the greatest obstacle to successful farming at this hour and by no means a trivial matter in public health—the two agencies upon which the power of a country must be built.

The veterinary profession needs the aid of all citizens in building up better and bigger veterinary colleges, more research work on animal ailments, and better conditions for the practitioners, who not only come to the aid of the public officials in conquering the more grave outbreaks of contagious plagues but who stand ready, day and night, to give medical attention to ailing live stock.

This, my friends of the radio audience, is the plea of the veterinary profession to the people of our great country and especially to those of the great livestock basin stretching in every direction from this important industrial center.

*Excerpts from a radio address sponsored by the Committee on Public Relations of the American Veterinary Medical Association at the 74th annual meeting. Similar addresses drawing attention to the importance of a competent veterinary service will be made preceding and during the 76th annual meeting.

Animal Health

ONE OF the principal duties of the veterinary profession is to inculcate a more general understanding of man's obligation to animals, particularly in the matter of preserving their health, which is equivalent to preserving the animals themselves. Without good health there can not be a good animal industry.

As stated editorially in *The Veterinary Record* (June 10, 1939), "In the animal world, where the intellectual pursuits do not enter, health and efficiency are virtually inseparable. The purpose of domestication of live stock is to subordinate them and their products to the needs of man, so when we direct our energies to securing the health of animals as an end in itself, we are serving humanity."

The veterinary profession, up to the present time, has been limiting its propaganda to the narrow field of transmissible disease, in the effort to attract public attention to its proper place in the social arena and, in doing so, it has failed to cultivate an understanding of its main purpose—the conservation of animal health, which is the undertaking that actually determines the welfare of mankind.

Is It Food or Medicine?

SKEPTICAL as the doctor and druggist may be, the day has arrived for quarreling over the question of what's what in food and drugs. The quarrel is already on between grocer and druggist. The dispute is mainly over vitamins, between the Retail Druggists Association and the IGA. When a prominent chain store company and, later, the IGA came out with a line of vitamins, the druggists held a mass meeting in Chicago to protest against the intrusion. The disputed merchandise included capsules combining all the known vitamins, as well as separate vitamins and various combinations, which the wisest doctor would hardly know for what sort of ailment these products should be prescribed.

But the sailing is not too smooth. Here and there the grocers are running into trouble. The Indiana Board of Pharmacy, for example, ruled that the grocers would have to get drug store licenses or quit selling vitamins, whereupon a chain store company filed suit protesting against the Board's ruling on the grounds that their stores have as much right to sell vitamins as vitaminized foods. And there, for the time being, the matter rests.

The situation is comparable to that created by wheat germ oil, which is widely sold through feed manufacturers and dealers, notwithstanding that the most astute veterinarians feel unequal to the task of selecting indications with satisfactory precision until a delicate test for vitamin E privation is determined. By the same token, it is logical to question the promiscuous use of thiamin chloride, nicotinic acid and other synthesized vitamin fractions, which have such tremendous value in the practice of medicine that every cook feels competent to recommend their use *ad libitum* in man and animals.

Vitaminotherapy is a science too far removed from the goal of an exact science to be turned over even to the hands of the trained druggist. It should certainly not be allowed to fall into the hands of the grocer and feed dealer without first obtaining the approval of the physician and veterinarian, respectively. This we say in the interest of all concerned: the manufacturer, the dealer, the druggist, the doctor, and the consumer. It is the art of diagnostics that, in the end, will promote the vitamin making to a stable industry.

Sir Frederick's Grief

RECALLING Frederick Hobday, the young, aggressive surgeon who acquitted himself as the master of his art at the clinic of the Association at Toronto in 1911 and who, through the years, became known to us for his illustrious career as journalist, teacher and clinician, in peace and in war, we in America will be pardoned for wondering just why the newspapers of London found cause to attribute the death of Sir Frederick to a "broken heart."

The press reports declare that the board of governors removed him from the council of the college for "lack of administrative ability," notwithstanding that somehow he managed to mobilize enough of that sort of ability to raise $500,000 to rehabilitate a tumbled-down institution which his successors now point out for its palatine dignity.

We were impressed with Sir Frederick's achievement of transforming the Royal College at Camden Town from "barn to palace" by years of tireless effort in behalf of veterinary education—to be rude but practical, by collecting sufficient money to do the job of restoring the first veterinary college in an English-speaking country to a state of physical respectability, a college which must have been dear to him.

American veterinarians who have followed his brilliant career through four decennia were ill-prepared for the sad news of the unhappy ending signalized in the London press. The kind of mundane strife that could torture a brilliant mind to that degree at the nightfall of a long and useful life is difficult to comprehend *outre mer*. We want to remember Sir Frederick by what he was, not by the sad obituaries sent to our desks by many of his admirers.

APPORTIONMENT OF AREAS FOR A.V.M.A. CONVENTIONS

Every other year, beginning with 1941, the annual meeting of the Association will be held in area 1. The conventions of the even-numbered years will be held in areas 4, 3, and 2, respectively. Thus, the 1942 meeting will be held in area 4; 1944, area 3; 1946, area 2. (This arrangement is subject to the approval of the House of Representatives at the Memphis meeting.)

The Future Veterinary Service

AS RAPIDLY as possible, the veterinary service should be developed into a disease-fighting army composed of a part-time personnel (practitioners) thoroughly disciplined and commanded by district officers. The reasons why this should be done are that such a service would be (1) economical and (2) efficient and sufficient.

In an address before the National Nutritional Conference, sponsored by the British Medical Association, Wm. C. Miller of the Royal Veterinary College (London) said in part:

> The health of the public in Britain is safeguarded to a very considerable extent by the control of certain diseases of animals exercised by the veterinary staff of the Health of Animals Division of the Ministry of Agriculture and Fisheries, together with a panel of part-time officers, chosen from among the veterinary practitioners, who perform their local duties under the direction of area officials.

In an article on the control of Bang's disease, R. R. Birch (*The Cornell Veterinarian*, April, 1939) wrote:

> It is well known that the use of the private practitioner to the fullest possible extent in doing the blood testing provides a maximum of efficiency and economy. . . . He is familiar with the herd and the problems of the owner.

At the 12th International Veterinary Congress (New York, 1934), Leclainche pointed out that the private veterinarian is the true livestock sanitarian because he knows the man, the farm and the disease problems.

This setup, although already partly in operation in this country, would furnish our livestock industry with disease-fighting machinery of the highest order and at a cost that would be very low compared with the tremendous amount of property that would be saved.

The future activities of organized veterinary medicine should be planned with this desirable objective in mind. The plan is now and has been on the agenda of the Committee on Public Relations awaiting definite action. A larger membership constantly enlightened on such matters in local, state and national association work could soon bring such reforms to a head.

The Origin of Meat Inspection

TO STUDY the origin of meat inspection one does not begin with Ostertag or the French regulations of the twelfth century. Better to turn the pages of history back about 30 centuries and leaf through the Talmud, the Holy Scriptures, Plutarch Lives and biography of the Pharaohs. The history of the Children of Israel in Egypt is a good starting point. These historic tribes lived under the Pharaohs for two or more centuries before Moses was rescued in the bulrushes.

Knowledge of medicine, perhaps less sketchy than is generally supposed, was a part of the Egyptian educational curriculum. The Jews were herdsmen, pastoral nomads, who understood the principles of livestock sanitation which still govern our task. When anthrax (?) broke out among their herds, these mahatmas moved their flocks from the infected valleys to the rolling land of Goshen. Cleanliness, superlatively scrupulous, was an inviolable law for man, for animal, for material.

As all men know, Moses was educated by the Pharaoh. Under that tutorage he developed his flair for statecraft, government, community discipline and, above all, for the importance of wild and domestic animals as a means of self-contained existence for his people. Unfit food was proscribed in the interest of tribal health. Nothing of a sanitary nature was overlooked in the written code. Watchful eyes observed the transmission of disease from animals to man. To enforce preventive measures he wisely resorted to the medical customs of his day—religion. "For the Lord thy God walketh in the midst of thy camp, therefore shall it be immaculate" may be set down as the first sanitary measure of record, and of animals brought to the abattoir (tabernacle) for slaughter, "They shall be without blemish" is certainly the first meat-inspection regulation.

APPLICATIONS

As the Association convenes in Memphis, it has every reason to reflect with satisfaction on its progress in the year just past and to plan with enthusiasm for the year ahead. During the past six months alone, more than 600 new members have entered the A.V.M.A. and, from all indications, this record-making enrollment will continue. On the occasion of the 76th annual meeting, the officers of the Association express their appreciation of the spirited coöperation on the part of the deans of the colleges, the resident secretaries and the membership in general.

First Listing*

ADLER, DANIEL A.
821 W. Milford St., Glendale, Calif.
B. S., D. V. M., State College of Washington, 1931. Vouchers: G. W. Blanche and W. L. Curtis.

ARNOLD, JOHN J.
418 S. 15th St., New Castle, Ind.
D. V. M., Michigan State College, 1937. Vouchers: E. B. Ingmand and L. A. Merillat.

ARRINGTON, MIKE A.
Forrest City, Ark.
D. V. M., Kansas City Veterinary College, 1914. Vouchers: Clyde W. Denman and John H. Gillmann.

BALLE, EDWIN G.
8642-56th Ave., Elmhurst, N. Y.
D. V. M., Michigan State College, 1939. Vouchers: C. V. Clark and B. J. Killham.

BAUER, CHARLES F. W.
Florissant, Mo.
V. S., Ontario Veterinary College, 1903. Vouchers: S. W. Haigler and H. S. Richards.

BERRY, JOHN WILLIAM
Pulaski, Tenn.
D. V. M., Alabama Polytechnic Institute, 1923. Vouchers: John H. Gillmann and E. B. Mount.

BOEN, NELS THEODORE
Harmony, Minn.
D. V. M., Kansas City Veterinary College, 1915. Vouchers: Orval C. Selby and P. H. Riede.

BISSELL, ROY C.
Postal, N. Dak.
D. V. M., McKillip Veterinary College, 1916. Vouchers: Frederick Low and Fred C. Driver.

*See January, 1939, JOURNAL.

BRADLEY, WALTER B.
Caledonia, Mich.
D. V. M., Michigan State College, 1938. Vouchers: C. F. Clark and E. T. Hallman.

BRENNER, CARL F.
330 Marshall Ave., Webster Groves, Mo.
D. V. M., Iowa State College, 1935. Vouchers: S. W. Haigler and L. A. Merillat.

BUSIC, WILLIAM H.
3653—9th Ave., Los Angeles, Calif.
D. V. M., Ohio State University, 1939. Vouchers: W. L. Curtis and M. M. Johnson.

COCHRAN, WILLARD NELSON
Route 2, Flat Rock, Ill.
D. V. M., Terre Haute Veterinary College, 1912. Vouchers: J. H. Murphy and W. B. Holmes.

COFFEE, W. M.
La Center, Ky.
D. V. M., Indiana Veterinary College, 1918. Vouchers: D. E. Westmorland and A. V. Barnes.

CONGER, CLIFFORD V.
Kahoka, Mo.
D. V. M., Kansas State College, 1929. Vouchers: H. E. Curry and L. A. Merillat.

CREIGHTON, STERLING H.
Coffey, Mo.
V. S., Ontario Veterinary College, 1895. Vouchers: H. E. Curry and L. A. Merillat.

DAVIS, ELMER NELSON
221 E. 74th St., Kansas City, Mo.
D. V. M., Kansas City Veterinary College, 1916. Vouchers: H. W. Young and L. A. Merillat.

EARL, JOHN WYCKOFF
Hillside Ave., Bedminster, N. J.
D. V. M., Cornell University, 1938. Vouchers: Harry C. Millar and J. A. S. Millar.

EVELETH, DONALD F.
Veterinary Research Institute, Iowa State College, Ames, Iowa.
D. V. M., Iowa State College, 1934. Vouchers: Chas. Murray and H. D. Bergman.

FAVARA, JOHN BOYD
Greenville, Miss.
D. V. M., Alabama Polytechnic Institute, 1929. Vouchers: E. S. Brashier and O. M. Norton.

FAWCETT, JEFFERSON FRED
Princeton, Ill.
M. D. C., Chicago Veterinary College, 1905. Vouchers: L. A. Merillat and E. B. Ingmand.

FEINBERG, JOSEPH GEORGE
2632 Troost Ave., Kansas City, Mo.
D. V. M., Kansas State College, 1938. Vouchers: G. G. Graham and S. J. Schilling.

FESLER, JAMES R.
52 Court St., Janesville, Wis.
M. D. C., Chicago Veterinary College, 1908. Vouchers: A. V. Hollister and H. E. Mullen.

FIKE, HOWARD SAMUEL
Medina, Mich.
D. V. M., Michigan State College, 1939. Vouchers: C. F. Clark and B. J. Killham.

FLANIGAN, J. A.
Eugene, Ore.
D. V. M., Washington State College, 1920. Vouchers: Sherman L. Brown and Chas. H. Seagraves.

GRINER, ADLAI BEE
Box 203, Fitzgerald, Ga.
D. V. M., University of Georgia, 1926. Vouchers: Chas. C. Rife and L. A. Merillat.

GROSSMAN, HENRY EARLE
418 E. 23rd St., Brooklyn, N. Y.
D. V. M., Cornell University, 1938. Vouchers: W. A. Hagan and James R. Kinney.

GUTHRIE, CHESTER EARLE
Silvermine, Norwalk, Conn.
D. V. M., Iowa State College, 1938. Vouchers: M. Ray Powers and Geo. E. Corwin.

HECTORNE, RONALD LESTER
37 Lake St., Bridgeton, N. J.
D. V. M., Ohio State University, 1928. Vouchers: I. H. Shaw and John D. Reardon.

HERZER, PHILIP CHARLES
816 S. San Pedro, Los Angeles, Calif.
D. V. M., Cornell University, 1917. Vouchers: W. L. Curtis and John L. Tyler.

HIRSCHEY, WILBUR C.
1216 North Eaton St., Albion, Mich.
D. V. M., Michigan State College, 1938. Vouchers: C. F. Clark and B. J. Killham.

HUGHES, LYNN B.
Box 81, Waskom, Texas.
D. V. M., Texas A. & M. College, 1939. Vouchers: R. P. Marsteller and L. A. Merillat.

IMMENSCHUH, ALDIE PHILIP
Route 2, Box 631, San Diego, Calif.
D. V. M., Kansas State College, 1914. Vouchers: W. L. Curtis and John L. Tyler.

ISBELL, ROBERTA GEORGE
Box 323, Gadsden, Ala.
D. V. M., Alabama Polytechnic Institute, 1926. Vouchers: R. L. Mundhenk and L. E. Starr.

JOHNSON, HARRY PAUL
332 N. Harrison, East Lansing, Mich.
D. V. M., Michigan State College, 1939. Vouchers: C. F. Clark and B. J. Killham.

JOHNSON, NORMAN EDWARD
520½ S. Virgil St., Los Angeles, Calif.
B. S., D. V. M., Washington State College, 1938. Vouchers: W. L. Curtis and G. W. Closson.

JONES, FREDERICK CLIFFORD
Macomb, Ill.
D. V. M., Ohio State University, 1913. Vouchers: John D. Reardon and L. A. Merillat.

KELLY, STEPHEN G. C.
1201 S. A St., Elwood, Ind.
D. V. M., Indiana Veterinary College, 1906. Vouchers: J. L. Axby and L. A. Merillat.

LEONARD, ELLIS P.
Box 432, Summit, N. J.
D. V. M., Cornell University, 1928. Vouchers: C. P. Zepp and J. B. Engle.

MAJOR, FRED J.
Box 274, College Station, Texas.
D. V. M., Texas A. & M. College, 1939. Vouchers: R. P. Marsteller and L. A. Merillat.

MAYO, ROBERT HENRY
Forest, Miss.
D. V. M., Alabama Polytechnic Institute, 1939. Vouchers: R. L. Mundhenk and H. L. Rubin.

MCCOY, EDWARD JOSEPH
Hickory, N. Car.
D. V. M., U. S. College of Veterinary Surgeons, 1917. Vouchers: J. H. Brown and L. A. Merillat.

MEISNER, FRANK COLUMBUS
1035 Marietta St. N. W., Atlanta, Ga.
D. V. M., McKillip Veterinary College, 1916. Vouchers: J. L. Hopping and Lowell J. Kepp.

MINER, NATHAN
3437 Fulton, Brooklyn, N. Y.
D. V. M., Michigan State College, 1939. Vouchers: C. F. Clark and B. J. Killham.

MINGLE, CARROLL KIRKHAM
520 Hunter Ave., College Park, Md.
D. V. M., Ohio State University, 1930. Vouchers: Adolph Eichhorn and William M. Mohler.

MOORE, HUBERT OTIS
203 Hemphill St., Hattiesburg, Miss.
D. V. M., Indiana Veterinary College, 1907. Vouchers: E. S. Brashier and L. A. Merillat.

MOORE, JOHN HERBERT
22 E. Junipero St., Santa Barbara, Calif.
D. V. M., Chicago Veterinary College, 1915. Vouchers: W. L. Curtis and John L. Tyler.

MOORE, JULES VERNE
Hayt, Mo.
D. V. M., Cincinnati Veterinary College, 1918. Vouchers: H. E. Curry and L. A. Merillat.

MORRIS, ROBIN ROY
Room 6, War Memorial Bldg., Nashville, Tenn.
D. V. M., Terre Haute Veterinary College, 1914. Vouchers: E. E. Nisbett and John H. Gillmann.

MOSES, CLYDE R.
General Delivery, Lucedale, Miss.
D. V. M., Alabama Polytechnic Institute, 1937. Vouchers: Hartnell Robbins and T. W. Boman.

MURPHY, J. T.
Cuthbert, Ga.
D. V. M., Alabama Polytechnic Institute, 1936. Vouchers: J. L. Hopping and L. A. Merillat.

NELSON, NORMAN M.
416 Grove St., East Lansing, Mich.
D. V. M., Iowa State College, 1934. Vouchers: C. A. Brandly and H. D. Bergman.

OLIVER, GARNET HUNTER
1310 Acoma St., Denver, Colo.
D. V. M., Chicago Veterinary College, 1912. Vouchers: James Farquharson and I. E. Newsom.

OOSTEN, ROBERT Y.
Demotte, Ind.
D. V. M., McKillip Veterinary College, 1916. Vouchers: C. C. Donelson and J. L. Axby.

OZIER, TERRY S.
320 Moody Ave., Martin, Tenn.
D. V. M., Michigan State College, 1939. Vouchers: C. F. Clark and B. J. Killham.

ROSE, HAROLD
2734 Sedgwick Ave., New York, N. Y.
D. V. M., Michigan State College, 1939. Vouchers: C. F. Clark and B. J. Killham.

SEPPONEN, CARL M.
2500-16th St., San Francisco, Calif.
B. S., D. V. M., Washington State College, 1939. Vouchers: E. E. Wegner and J. E. McCoy.

SMITH, FLETCHER EUGENE
4801 Evanston Ave., Seattle, Wash.
D. V. M., Cornell University, 1905. Vouchers: R. A. Button and V. C. Pauhlman.

SPARHAWK, HOWARD HARVEY
701 Municipal Bldg., Akron, Ohio.
D. V. S., Ohio State University, 1907. Vouchers: A. J. De Fosset and O. V. Brumley.

VAN DE GRIFT, EDWARD RUSSEL, JR.
Box 723, Columbia, S. Car.
D. V. M., University of Georgia, 1930. Vouchers: R. A. Mays and Herbert Racoff.

VESTAL, HAROLD CLARK
313 N. W. Sixth Ave., Faribault, Minn.
D. V. M., Cornell University, 1915. Vouchers: B. S. Pomeroy and H. C. H. Kernkamp.

WAIND, WILLIAM GEORGE
McMinnville, Ore.
D. V. M., Ontario Veterinary College, 1912. Vouchers: Sherman L. Brown and Chas. H. Seagraves.

WEINER, MORRIS JOHN
4164 Drexel Blvd., Chicago, Ill.
V. M. D., University of Pennsylvania, 1938. Vouchers: Thomas E. Utley and H. E. Kingman, Jr.

WHITE, PAUL T.
1227 N. Capitol Ave., Indianapolis, Ind.
D. V. M., Ohio State University, 1933. Vouchers: Walter K. York and E. B. Ingmand.

WILSON, JESSE THOMAS
Box 23, Hampton, Va.
D. V. M., Kansas City Veterinary College, 1918. Vouchers: A. J. Sipos and L. A. Merillat.

WINTER, ALAN WILLIAM
205 E. Dewey St., Buchanan, Mich.
D. V. M., Michigan State College, 1938. Vouchers: C. F. Clark and B. J. Killham.

Second Listing

Bailey, O. Leighton, 10 S. Pine St., Spokane, Wash.
Baker, Harrison V., 161 Main St., Hamburg, N. Y.
Baker, Howard L., Jr., Centre Square, Pa.
Beers, Alfred M., 49 South, Warwick, N. Y.
Broadwell, John M., R. F. D., Box 162A, La Porte, Texas.
Canfield, Rolden F., 10 S. Pine St., Spokane, Wash.
Carter, Ocie, Box 317, Paris, Ky.
Chapman, Lee Roy, Box 411, Snyder, Texas.
Chapman, Merwyn P., 509-11 Federal Bldg., Indianapolis, Ind.
Christian, John R., Woodhull, Ill.
Claflin, Merle Lyman, Barron, Wis.
Coale, Andrew J., 115 E. Cherry St., Carmi, Ill.
Conboy, Joseph R., 364 S. Broadway, Yonkers, N. Y.
Donohue, Robert J., Toppenish, Wash.
Durner, Edward A., 453 E. 142nd St., New York, N. Y.
Edds, George T., Box 215, Faculty Exchange, College Station, Texas.
Farris, Merle LeRoy, Box 886, Kingsport, Tenn.
Field, Horace Candrian, 2050 N. Berendo St., Hollywood, Calif.
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Fohey, George James, Clio, Mich.
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The amount that should accompany an application filed this month is $6.67. This covers membership fee and dues to January 1, 1940, including subscription to the JOURNAL.

CLINICAL DATA

Primary Osteogenic Sarcoma of the Mammary Gland of a Dog

By CARL F. SCHLOTTHAUER,* D.V.M., and WARREN B. THURBER, D.V.M.
Rochester, Minn.

BENIGN osteomas and various neoplasms containing bone or calcified tissue are not infrequently observed in lower animals. These tumors, however, must not be confused with true osteogenic sarcoma. The adjective, osteogenic, refers to tissues arising from bone-producing cells. Meyerding[1] stated, "An osteogenic sarcoma is a malignant tumor which develops from tissue that is predestined to form bone." He further mentioned that many of the tumors previously designated as osteosarcoma, osteochondrofibrosarcoma, osteochondrofibromyxosarcoma, chondrosarcoma and periosteal osteogenic sarcoma are osteogenic sarcomas.

From a review of the literature dealing with neoplasms and from personal observations, we are of the opinion that osteogenic sarcomas are of infrequent occurrence in lower animals. In the course of necropsy performed on 768 horses, 1,548 dogs, 401 cats, 70 bovines and 44 hogs, Crocker[2] observed an osteosarcoma of the mandible in a horse, an osteosarcoma of the left humerus in a dog, and one of the left femur in one cat and a giant-cell osteosarcoma on the left foreleg of another cat. Stubbs[3] reported the occurrence of osteogenic chondrosarcoma on the proximal extremity of the left femur in a cat.

Feldman[4] observed one osteogenic sarcoma in his series of 81 tumors obtained from dogs. This occurred on the distal portion of the left ulna of a 2-year-old male English Setter. The neoplastic process later invaded the radius. Jackson[5] noted the occurrence of an osteogenic sarcoma on the humerus of a dog and on the hock of a cat. One of us (Schlotthauer[6]) reported the occurrence of an osteogenic sarcoma on the head of a dog. This neoplasm apparently had its origin in the ethmoid and frontal bones. It invaded the frontal and maxillary sinuses, the left orbit and the anterior portion of the cranial cavity, where it exerted pressure on the frontal portion of the brain and caused symptoms of brain injury.

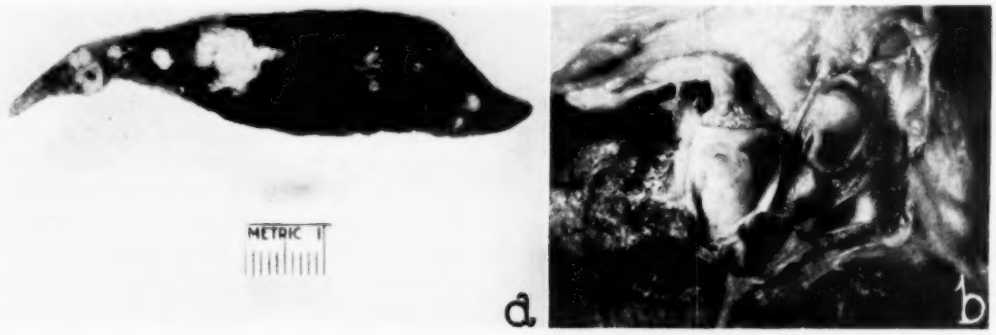

Fig. 1. Gross appearance of the metastatic tumor nodules (a) in the lungs; (b) protruding from the lumen of the pulmonary arteries.

*Division of experimental medicine, The Mayo Foundation.

At this time we wish to report a primary osteogenic sarcoma of the mammary gland in a 12-year-old dog. This neoplasm re-

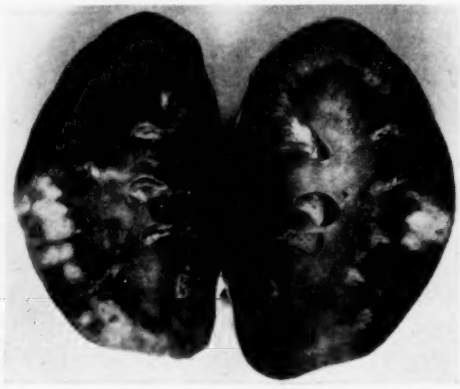

Fig. 2. Gross appearance and situation of the metastatic tumor nodules in the right kidney.

curred following its surgical removal and metastasis occurred in the right axillary lymph node, both lungs, the heart and the right kidney.

REPORT OF CASE

A 12-year-old mongrel Chow female dog was brought to us on February 8, 1939, because of a large tumor of the right third

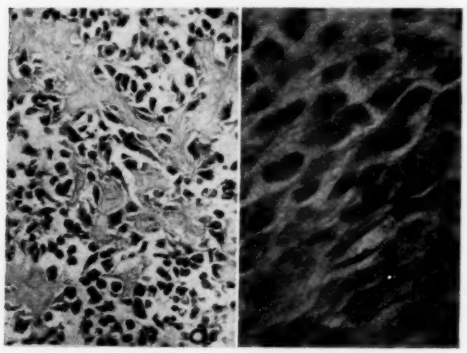

Fig. 3. (a) An abundance of osteoid tissue in the primary neoplasm (x 125); (b) the cell type of the neoplasm. Several cells are undergoing mitosis (x 340).

mammary gland. This tumor had been present for several weeks and had increased slowly but progressively in size. At the time it was approximately 10 cm. in diameter. Examination revealed it to be smooth but slightly irregular, very hard and freely movable. Metastasis could not be demonstrated by palpation or gross observation. Because of the hardness of the tumor and the fact that it appeared to be encapsulated, it was thought to be one of the comparatively benign, so-called mixed tumors that occur rather frequently in the mammary glands of dogs. Since surgical excision of these new growths usually results in a permanent cure, surgical treatment was advised in this case.

Because the dog was in rather poor physical condition, a minimum of dissection seemed desirable. Therefore, with the animal under general anesthesia the tumor and the affected third mammary gland only were excised asceptically. The surgical wound healed rapidly and the immediate results of the operation were satisfactory.

Scattered spicules of bone were encountered throughout the neoplasm when it was cut open for gross inspection. Microscopic study of tissue removed from various portions of the tumor revealed an osteogenic sarcoma. All of the sections were similar. The neoplasm was composed chiefly of osteoblasts, osseomucin and osseous tissue. Many of the small vessels were occluded with neoplastic tissue and many cells were undergoing mitosis. The neoplasm appeared to be of a rather high degree of malignancy and recurrence was anticipated.

The owner noted beginning recurrence of the neoplasm about the third week following surgical excision of the primary tumor. Because of the dog's poor physical condition and the apparent high degree of malignancy of the neoplasm, a second operation was not advised. On April 4, 1939, it was decided that the dog should be killed. At this time a large, irregular, hard but freely movable tumor approximately 10 cm. in diameter was present immediately anterior to the site of the original neoplasm. The dog appeared emaciated and weak and marked dyspnea was noted. Examination revealed gross evidence of metastasis. The right axillary lymph node was markedly enlarged and hard. Roentgenograms of the thorax revealed numerous metastatic tumors in the lungs. Euthanasia was accomplished by the intravenous administration of a lethal quantity of pentobarbital sodium (so-

dium ethyl [1-methylbutyl] barbiturate) and necropsy was performed.

The gross lesions present at necropsy were as follows: The right axillary lymph node was 3 by 5 cm. in diameter and it contained some bone tissue. Both lungs contained numerous metastatic nodules of varying size (fig. 1*a*). Cross section of the pulmonary arteries revealed the neoplasm to be growing within the lumen of the vessels (fig. 1*b*). The myocardium of the right ventricle contained several small metastatic nodules. Tumor tissue also surrounded some of the chordae tendineae, but did not appear to invade them. The right kidney contained numerous metastatic nodules (fig. 2) but the left kidney appeared grossly normal. The other abdominal or pelvic organs appeared to be essentially normal.

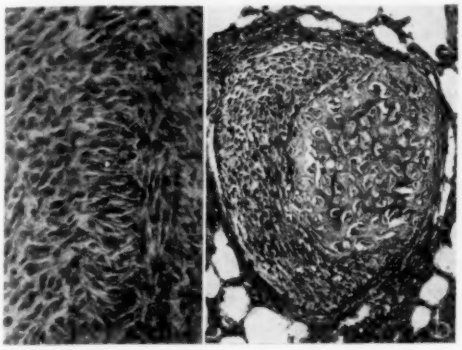

Fig. 4. (a) The elongated cells which resemble fibroblasts appear to be giving origin to osteoblasts (x 125); (b) a mass of metastatic osteoblasts and osteoid tissue within the lumen of a small pulmonary vessel (x 70).

Histologic study of the primary neoplasm, the recurrent tumor and the metastatic nodules revealed the cell type and histologic structure of all the growths to be similar. The primary neoplasm and the recurring neoplasm however, contained more adult osseous tissue than the metastatic nodules (fig. 3*a*). The cells in all of these neoplasms were chiefly sarcomatous osteoblasts. In some sections studied many of the cells were in a state of mitosis, indicating rapid growth (fig. 3*b*). According to Broders[7] and Feldman, these sarcomatous osteoblasts probably are the result of metaplasia of multipotent connective-tissue fibroblasts.

As osteoblasts, they acquired the capacity to produce osseomucin, in which lime salts are deposited to form adult osseous tissue. In some sections of the primary neoplasm this transformation of sarcomatous fibroblasts into sarcomatous osteoblasts is suggested strongly (fig. 4*a*) but the metastatic nodules were composed entirely of sarcomatous osteoblasts, osseomucin and osseous tissue. Many of the smaller vessels in the lungs were entirely filled with masses of this neoplastic tissue (fig. 4*b*).

COMMENT

Feldman mentioned that true bone tumors may arise occasionally in the soft tissues separate from the skeleton, but he did not cite any cases. Daille and Sabattie[8] reported the occurrence of an osteosarcoma in the testicular region, with generalization, in an ox castrated by the method of bistournage. This is the only report of a case of osteogenic sarcoma arising in the soft tissues of a lower animal that we found reported in the literature.

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Contrary to the popular belief, the bite of the tarantula is not poisonous—no more so than the prick of a pin.—*Natural History Magazine.*

Sulfanilamide Treatment of a Canine Hemolytic Streptococcemia Characterized by Edema of One Foot and Leg*

By R. F. LIGHT, B.S., M.S., and CHARLES N. FREY, B.S., M.S., Ph.D.
New York, N. Y.

DURING the past three years six of our Great Dane puppies, varying in age from 8 weeks to 6 months, have developed a characteristic syndrome which appears to be due to a hemolytic streptococcemia.

The symptomology of this disease is as follows: The first sign is a dull, listless condition, with a concomitant loss of appetite. There is a slight rise in temperature and in 24 to 48 hours, there is an edema of one paw, either the fore or hind quarter. This swelling is slight and, unless one is watching for it specifically, the swelling may be unnoticed for a day or so. In our initial examination of the swollen paw we were not able to detect a primary lesion or focus of infection in any of the six cases observed.

Fig. 1. Dog 51, showing edema of right forepaw characteristic of early stages of this disease. The dog had a temperature of 104.1° F. The condition responded promptly to sulfanilamide therapy.

As the disease progresses, the temperature rises to between 104° and 107° F. The edema of the foot increases and spreads to the leg. Eventually, the whole quarter is swollen and edematous, and the animal dies. At no time during the course of the disease did we find evidence of a pus pocket or a definite focus of infection. On autopsy we found no abscess.

The first animal which became infected was treated locally with camphorated oil and wet compresses of Burow's solution, and was given laxatives and methenamine. The dog survived twelve days after the symptoms had become acute, having a temperature of 105° to 107.5° F. for the last eight days.

The next year, when one of our animals showed the same symptoms, we treated the symptoms for two days, without favorable results, and then tried sulfanilamide therapy. There was an immediate response to this treatment. The protocol of this animal, No. 42, is given below:

October 14.—Dog 42, male, age 16 weeks, weight 35 pounds. Animal shows swelling of the left hind foot and leg. No sign of injury; no sensitive area; animal dull and listless; poor appetite; temperature, rectal, 105° F. Treatment: Castor oil, wet compresses of Burow's solution and two 5-gr. tablets of methenamine.

October 15.—No improvement; edema of foot and leg increased, extending up to hip; temperature, 104.8° F. Treatment: Wet compresses of Burow's solution and two 5-gr. tablets of methenamine.

October 16.—No improvement; temperature, 104.1° F. Treatment: Burow's solution compresses; two 5-gr. tablets of meth-

*From the Fleischmann Laboratories, Standard Brands Incorporated.

enamine; in the afternoon gave two 5-gr. tablets of sulfanilamide.

October 17.—Animal looks better; edema checked; temperature, 102.6° F.

October 18.—Temperature 101.1° F.; swelling subsiding; appetite good.

October 19.—Temperature, 101.1° F.

October 20.—Temperature, 101° F.; swelling almost gone.

October 22.—Animal normal.

The third dog to become infected was not treated until four days after edema of the left fore foot had been noted. On the fifth day a blood culture was taken and 20 gr. of sulfanilamide was administered.

In the case of the last three animals which showed these characteristic symptoms (swelling of the foot, loss of appetite, rise in temperature, etc.) we did not allow the disease to progress to the acute stages. Sulfanilamide was administered immediately (20 gr. in doses of 5 gr. every three hours). The temperature of the animals dropped at once, the edema was reduced in about 72 hours, and the animals returned to normal in three to four days. Figure 1 shows the fore paws of one of these dogs (No. 51, male; age 9 weeks; weight, 7 kilos).

The blood cultures of the second and third dogs to contract this disease were positive for a hemolytic streptococcus. Blood cultures of the last three dogs taken from the external saphenous vein were negative. This may have been due to the fact that the samples had been taken before the animals became acutely ill and the septicemia had not advanced sufficiently. Unfortunately, blood cultures of the first animal, which died, were not made.

CONCLUSION

We have described a cryptogenic septicemia in dogs due, we believe, to a hemolytic streptococcus. The symptoms are an edema of one quarter, starting with the foot and eventually involving the whole leg and shoulder or hip, a marked rise in temperature, loss of appetite, and other symptoms concomitant with high fever. The disease responds readily to sulfanilamide therapy.

Pathology of Demodex Infection

By EUGENE MAIER,* Ph.D.
Venice, Fla.

IN THE COURSE of studies on the infection of dogs with *Demodex folliculorum*, histologic sections were obtained which are herewith described.

The mites together with the ticks constitute the order of Acarina, small Arachnoidea, which are subdivided into a number

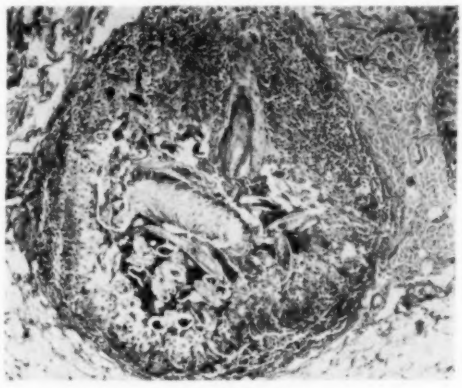

Fig. 1. This section shows a whole lesion with the hairshaft in the center. The most prominent feature is the circular wall around the lesion consisting of a rather heavy infiltration of round cells from without. The hair shaft in the center is cut tangentically and is in the process of autolysis. On top of the shaft a parasite is cut sagittally and below the shaft are two more parasites, cut in the same way. In the surrounding tissue adjacent to the shaft and within the wall of the lesion many parasites are seen, most of them cut transversely.

of families. The mites show a considerable number of habits. They are parasites of mammals and birds. Like the tick, many are external parasites, feeding on the blood of their hosts. Some, like the Sarcoptidae and Demodicidae, burrow in the skin of the host and cause severe itching and disease. They are parasitic in the hair follicles and sebaceous glands of man and animals.

The hair-follicle mites of different animals are difficult, if not impossible, to differentiate as distinct species. *D. canis* Leydig attacks dogs and its distribution is cosmopolitan. It causes follicular or red mange in the dog and has been reported as

*Florida Medical Center.

parasitic for man.* *D. cati* Megnin in the cat, *D. bovis* Stiles in cattle, *D. equi* Daillet in horses, and other species are formed on various animals. These parasites are also found in the hair follicles and sebaceous glands, the head toward the bottom of the sac and the caudal end toward the external opening. As many as 150 have been found in a sebaceous gland, but generally there are 30 to 50 of them.

The larvae and eggs, often in great numbers, lie beside the parasites. The irritation brought about by the presence of the parasite causes dilatation of the follicles and sebaceous glands, and the subsequent atrophy of the hair papillae results in loss of the hair.

*Todd and Sanford: Clinical Diagnosis by Laboratory Methods. (W. B. Saunders Co., Philadelphia, 1927.)

—"Dos and Don'ts"

(*Continued from page 350*)

We usually have one physician, one dentist and a professional man experienced in radiography who render their services to us without charge, but the accommodation is usually returned many fold. We do not hesitate to call our favorite physician on matters that need furthering in our field. Particularly in surgery he is of invaluable assistance. Our dentist gives us advice in local anesthesia, packing cavities, and selection of equipment.

A matter that confronts every practitioner is collections. The proper training of an assistant on collecting bills is important. A certain percentage of the people will call when they are positive you are not in the office, their idea being to save a few dollars or to inveigle your assistant into accepting the patient on credit. Allowing the assistant to be responsible for such cases soon remedies the situation. In country practice, become acquainted with every farmer. Thereby, you can soon learn the financial status of all potential clients.

Clients operating a business or living in the city respond well to statements, but the average farmer pays little attention to them. The majority of large animal practitioners send statements two to four times a year to certain clients. This proves that the farmer is still bent on being independent and pays as he wishes. Once a farmer owes you a bill and does not require your services any more, collect immediately. If he continues to call on an old bill, judicious handling will bring full payment in due time.

If in our practice we receive a call outside of our established territory, we first investigate the client's financial standing with other veterinarians. If he has not paid his bills, we do not hesitate to make the call, but merely inform him that until all back bills are paid, we shall not call again. In some cases, we have been instrumental in collecting all back bills for our colleagues.

By way of conclusion, I should like to offer this pithy reminder—the most important of all the "don'ts": "Don't look to display advertisements in telephone directories and newspapers for your success as a practitioner. The doctor who advertises not only loses the respect of his colleagues and, subsequently, of his clientele but also serves to break down the reputation that the profession as a whole is slowly yet definitely building up.

Ether Anesthesia

Ether anesthesia induced by the open drop method is the best method of general anesthesia in the hands of the average person. Not infrequently, it should be resorted to even by expert anesthetists, when the results they are attempting to obtain by some other method of anesthesia are unsatisfactory from the standpoint of relaxation and from that of quiet respiration.—*John S. Lundy in Surgery, Gynecology and Obstetrics, lxvi, May, 1938, pp. 935-936.*

One of a pair of prized twin lambs was lost to a farmer of Coldwater, Mich., recently, when it became overinquisitive about a goose sitting on her nest. The goose battered the lamb to death with its beak and wings.

Skeletal Diseases of the Horse

By H. C. CRAWFORD, V.M.D.
Jamaica, Long Island, N. Y.

AUTHORITIES in equine medicine regard osteomalacia as the principal bone disease of mature horses and rachitis, or osteolymphatisme, as the dominant skeletal affection of colts.

In 1897, Georges Joly of the French Cavalry School at Saumur[1] hypothesized that *osteitisme* is the major cause of equine skeletal derangements. The observations of other workers, notably Lienaux of Brussels[2] and Kitt of Munich[3] (who termed the condition *ostetismus universalis*), verify Joly's deduction. Cadiot and Almy,[4] Curot,[5] and Gobert and Cagny[6] regarded *osteitisme* as an industrial malady.

A malady characterized by a demineralization syndrome has been recognized since about 1900 by the veterinarians of Russia and the Latin and Teutonic countries as the agent responsible for most of the cases of halts of locomotion due to a skeletal pathogenesis.

The writer is reasonably confident that the majority of cases designated by English-speaking veterinarians as osteomalacia are, in reality, *osteitisme* Joly or *ostetismus universalis* Kitt.

Demineralization of the horse may be manifested as a distortion, subluxation, ligamentous or tendonous dysinsertion, cartilaginous erosion, osseous vegetation, osteoarthritis, or spontaneous fracture. In fact, all cases of foot lameness which are characterized by a primary osteitis of the os pedis and a secondary podophyllitis chronica are *osteitisme* Joly.

The rôle played by avitaminoses as a contributory factor in the skeletal pathology of the adult horse is not established. However, it is definitely known to be a contributory agent in rachitis.

Allied with intensive agriculture, the feeding of proprietary mineral supplements, pasture rotation practices, and intensive breeding problems, wherein all too frequently the animals are underfed or, for the sake of economy, inferior feeds are provided, are an increasing number of cases of demineralization in foals *in utero*.

It has been known for some time that when a horse shows clinical evidence of one of the spavin group of lamenesses, the condition is due to some hereditary predisposition or a skeletal diathesis. It has been definitely shown, however, that such hereditary traits can be eradicated through the proper allotment of minerals. In one instance, an entire stud whose offspring were thus affected was moved to a mineral-efficient location, with the result that all of the foals born in this new location were free of such defects.

Often, a mare that has been utilized in racing or some other commercial pursuit is retired to a stud where there is a mineral deficiency of the soil or drinking water, or both. She receives her nutritional requirements from grass, grain and hay produced on this soil and, subsequently, her foal *in utero* becomes a victim of demineralization. After her offspring has been broken, it is probably given drastic vermifuge treatments and then rushed into training. Resultant, the inroads of fatigue produce a disturbed mineral metabolism which manifests itself as the *osteitis of fatigue, osteitisme*, or *ostetismus universalis*.

The *osteitis of fatigue* accounts for as much as 50 per cent of the failures in roaring operations. Such failures are not due to unskilled surgery but, rather, to the osteodystrophy of the larynx (which may or may not have been detectable on palpation) that antedated the operation.

Calcification of the larynx does not necessarily have to reach the progressive stage in order to be detected on palpation but, nevertheless, osteodystrophy of that structure is frequently present in conjunction with the neuropathic phenomenon.

Relative to the application of firing as an intervention to correct osteoarthritis, the

practitioner is confronted by the limited response to such treatment for an osteitis systemic in character (carpitis, tarsitis, sesamoiditis) as compared to one local in type.

Systemic osteitis only rarely responds to firing, but an osteoperiostitis of a local type may respond within certain limits, *i.e.*, reduction in size of exostoses as well as checking the productive process.

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Comment: Although the question of osseous vegetations on the legs (foot) of horses remains controversial, opinion leans mostly toward the rarefied state of the equine skeleton as the etiological factor. Recent work of German authors, which conflicts with that of Mitchell *et al*, who for want of a better name classify these bony vegetations under the general head of "rheumatic disease," are by no means convincing, in the face of the indisputable fact that the bones of the domestic horse are fragile and the fusing of the ligaments to them is so insecure as to leave the skeleton and ligaments vulnerable to stress.

In this field of investigation the observations of Mitchell, Kinter, Holt and Greenlee are too outstanding to be set aside as inconsequential. It now seems quite evident that when the precise nature of these osteopathic infirmities, so peculiar to the horse, is clarified, the basis of their pathogenesis will be a structural deficiency of the skeleton. At least, the teachings of modern physiology all point in that direction.

Dentigerous Cyst

To the Editor.—A few months ago I removed a cyst containing three tooth particles from the temporal region of a horse. The whole mass was the size of a hen's egg. The three particles overlapped one another and I thought that everything had been removed. Except for drainage, the wound was closed with sutures. The cavity has not healed and is now filled with wart-like granulations, which the horse rubs as if the formative tissue were itchy, and causes it to bleed.

Would you advise another operation to search for some remaining particles, or is there any local application that might hasten cicatrization? The granulations extend beyond the level of the region. Its surface is raw and 1½ inches in diameter. —*C. C. H., D.V.M., Illinois.*

Reply.—In the removal of a so-called dentigerous cyst, which is a common congenital aberration of horses and small ruminants, the main pitfall is failure to remove the entire content of the cavity. Obviously, in this incident a remaining tooth particle is obstructing cicatrization of the surgical wound. The entire granulating mass should be scooped out and the offending particle removed. Local application will not be of any aid.

Our World War Horses, Overseas

By combing through the records of the remount and veterinary services, the authors of "Veterinary Military History of the United States" set the number of horses and mules used and in use overseas up to the time of the Armistice at 173,482.* A new book, "American Armies and Battlefields in Europe," by General John G. Pershing, gives the number as 243,039 for the entire period of the war and occupation of German territory following the cessation of hostilities.

The cost of these animals per head was $416.63. They were purchased from the French and British, this new book says. Up to the Armistice, only 2,917† came direct from the United States.

In comparing hog-cholera virus of Brazil with that of Algeria, Portugal, Argentine and the United States, it was found that the viruses obtained from these various sources differed only in virulence. (*A. Braga, Rev. do Dep. Nac. du Prod. Ani, Abs., Rev. de Méd. Vét., xc, Sept., 1938, p. 525).*

*New Bulletin, Remount Service, Dec. 18, 1918.
†Veterinary Military History of the United States, Vol. II, p. 671.

Pyosalpinx in a Holstein Cow

By J. F. BULLARD,* D.V.M.
Lafayette, Ind.

PYOSALPINGITIS, or pyosalpinx, in dairy cows is not uncommon, although it does not occur as frequently as many other conditions affecting the genital tract. It is not diagnosed easily and, therefore, we do not suspect it as often as endometritis, cervicitis, or various ovarian dysfunctions. When these can be reasonably eliminated and sterility persists, we are then justified in at least suspecting some type of inflammatory condition in the fallopian tubes, even though in many instances they may not be accurately palpated by rectal examination unless distinctly enlarged due to chronic inflammatory processes.

Various forms of salpingitis are definite causes of sterility. With this in mind, we would naturally suspect that the case to be discussed started after the last conception had occurred.

The following clinical history briefly gives the outstanding facts regarding the case.

The subject, a Holstein cow, was born on July 22, 1934. On November 3, 1936, the animal freshened. On February 3, 1938, the cow freshened and retained placenta. The following day, an examination was made and the placenta removed.

It is the practice in this herd, when the animals are apparently normal with the exception of the retained placenta, to wait for 24 hours and then make an examination. Inspection, after removal, revealed the fetal membranes were apparently all present. Four No. 10 gelatin capsules of boric acid and azamine were inserted deep into the uterus (5 gm. of azamine and 8 ounces of boric acid).

On February 5, 1938, four capsules of azamine and boric acid were given. Vaginal examination two days later revealed that the condition was progressing satisfactorily.

A very severe lameness developed in the left hind leg on February 9, making it nearly impossible for the animal to stand. When this cow was forced to move, she did so very reluctantly and showed severe pain. Due to the fact that on previous examinations the uterus had been involuting satisfactorily, no examination was made at this time. It was suspected that an injury had occurred while the animal was getting up or laying down. However, the difference in the alignment of the hips which occurs in dislocations was not present and there was no injury to the external angle of the ilium. Further, the condition was not characteristic of obturator and paralysis. From the date of calving this cow remained in a box stall and every comfort was provided for her.

Since the condition still persisted on February 12, a careful rectal examination was made. The uterus, although naturally not completely involuted, could be palpated in its entirety. In the region of the left ovary there was a large, round, smooth surfaced, fluctuating, very sensitive mass about the size of a grapefruit, apparently attached to the anterior end of the left uterine horn.

Observations on February 17 and 19 showed the condition to be the same. Although still slightly painful to palpation on March 1, the mass had become smaller and was now about the size and shape of a hen's egg. From February 19-28 this individual had gradually improved and, at the present examination, she moved and walked in a perfectly normal manner. The herdsman was advised to give this cow complete sexual rest for several months.

On September 16 the herdsman reported that this cow had been in estrum quite a few times during the late spring and summer. Rectal examination revealed the uterus to be pelvic in position. A corpus luteum was expressed from the right ovary. The left horn was very tense and stretched,

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due to severe adhesions on its anterior end to the wall of the rumen. At this point the same egg-sized mass could be felt. There was no evidence of pain. The left ovary could not be recognized. Slaughter was recommended.

Rectal palpation on February 7, 1939, revealed conditions similar to those observed in the examination of September 16, 1938. The vaginal mucus was clear. Again it was recommended that this cow be slaughtered.

The animal was slaughtered on May 5. A small, very hard, round, fibrotic mass was found which caused the left horn to adhere to the rumen wall. This was carefully dissected from the rumen and the entire genital tract removed. The mass measured approximately 2 inches in diameter and was apparently an enlargement of the fallopian tube at its anterior or ovarian end. When incised, about 1 ounce of thick, yellowish pus oozed out. The capsule was three-eighths of an inch thick. By careful probing from the lumen of the tube, distal to the mass, a small opening into this abscessed area was found. The left ovary was very small and fibrotic and closely imbedded in the connective tissue adhesions surrounding the abscessed tube. A slight mucopurulent exudate was found in the anterior portion of the left horn and slight evidence of endometritis was observed. The right ovary contained a well formed corpus luteum. The external *os uteri* showed a very slight congestion and the vagina appeared normal. In the cervical canal, there was a trace of mucopurulent exudate.

The outstanding factor in this case was the apparent rapidity with which this abscess developed. Although during the course of treatment for retained placenta this area was not palpated, one would be reasonably safe in surmising that the abscess was not present at the time of parturition. When first found, it was in an acute stage, as evidenced by its size and sensitivity. It is improbable that it was present at the time of the second conception, for, if it had been present, pregnancy more than likely would not have occurred. It appears to have resulted from an ascending infection as a sequel of retained placenta.

Alcoholic Intoxication in Swine

By W. E. MARTIN, JR.*
Wapakoneta, Ohio

ON THE MORNING of July 18, 1939, the writer accompanied his father, a general practitioner, to a hog-feeding farm where garbage was the main sustenance.

One entire section of the herd, consisting of fourteen 80-pound shoats and a sow, was affected. The general appearance was that of a cholera-infected herd. All of the animals were in a semi-comatose condition. When prodded with the toe of a boot, some of them could manage to get to their feet and stagger about the lot, with the posterior quarters swaying and stepping extremely high.

Inquiry into the feed revealed that stale bread had been soaked in a water barrel left standing in the heat of the July sun for about two weeks and had been fed for the first time three hours before our arrival. Investigation of the barrel revealed an odor suggestive of alcohol and the breath of the hogs also revealed traces of alcohol. The owner stated that all of the hogs had appeared normal at the morning feed and had become affected three hours later. Suspicion of an infectious disease was excluded by reason of the sudden onset together with the slightly subnormal temperature (100.3° F.).

Each hog was given 3 minims of fluid extract of nux vomica by mouth. The writer returned to see the herd at six that evening. All of the shoats and the sow had made a complete recovery.

Transplacental infection of swine erysipelas was observed by G. Comotti (*La Clinica veterinaria*, July, 1938) when on the death of a sow and her 4-day-old litter, the specific organism was found in the sow and in three out of five of the dead pigs. The sow showed the classical lesions (endocarditis) of the disease post mortem but in the pigs only congestive lesions of the kidneys and lungs were found.

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*Ohio State University, class of '40.

Poisoning by Hydrocyanic Acid

By E. C. W. SCHUBEL, D.V.S.
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THE AGRICULTURAL experiment station of the University of Wisconsin has investigated the problem of Sudan grass poisoning and offered some very valuable suggestions.

It was found that the short, dark green Sudan grass is high in cyanide poison and is dangerous to pasture. Second growth after pasturing or removal of a hay crop, when short and dark green, is especially dangerous. Sudan grass which is 2 feet or more in height, whether first or second growth, is usually low in cyanide poison and is relatively safe to pasture. Sudan grass, short or tall, which is of a pale or yellowish green color is low in cyanide poison and relatively safe to pasture. Dried and browned leaves and tips of leaves should not be mistaken for a pale or yellowish-green color, for often among the short, dark green plants—high in cyanide poison and dangerous—there are present, due to drouth, many dried leaves of this kind.

Both from the standpoint of the danger of poisoning and the possibility of obtaining the most pasture, Sudan grass usually should not be pastured until it has reached a height of at least 12 inches, and preferably 24 to 36 inches. Many fields of Sudan grass are uneven or spotted. Every field, before pasturing, should be gone over carefully and, if there is any doubt, samples should be taken and tested before turning in the cattle.

A high level of available nitrogen and a low level of available phosphorus in the soil tend to increase the poison content of Sudan grass, while a low level of available nitrogen and a high level of available phosphorus have the opposite effect. A high cyanide content, however, may still occur in short plants, especially in the second growth, even though the level of available phosphorus is high. A high level of available phosphorus along with the other favorable growth factors make it possible for the plant to attain quickly a growth of 2 to 3 feet, at which height it is relatively free of poison.

Drouth tends to keep the plants small, and in this stage they are always much higher in cyanide than when larger. Drouth retards growth by withholding water and probably lessening the availability of phosphates to plants much more than that of nitrogen.

When Sudan grass is dried and made into hay, the cyanide-poison content does not change greatly. Since it is usually not cut for hay until it reaches a height of 3 feet or more, there should be little, if any, danger of cyanide poison in Sudan grass hay. However, if short Sudan grass is made into hay, it will be dangerous as a feed.

Cattle, when turned into Sudan grass of high poison content, usually stop eating after 15 minutes, due to the action of the poison. If the animals are not too hungry and if they are in a high state of vigor, they usually stop eating before they take a fatal dose. The amount of cyanide poison that will prove fatal varies with each animal. If the cattle are in a low state of vigor and very hungry, they are more apt to eat a fatal dose than when the opposite is the case. As an added precaution, it is a good plan to watch the herd for an hour when it is first turned into a field of Sudan grass. If some of the animals stop eating or look around for other grass to eat, it is a good sign that the Sudan grass is dangerous.

A great many other plants are capable of developing hydrocyanic acid and may be responsible for poisoning. Among such plants are the following:

Choke-cherry (wild).
Black cherry (wild).
Sorghum.
Johnson grass.
Flax.
Arrow grass.
Velvet grass.
Christmasberry.

Hydrocyanic acid is not found in any appreciable quantities in healthy, growing plants. The acid develops only when normal growth has been retarded or stopped by drouth, frost, bruising, trampling, wilting, mowing, or other causes. Under such conditions, hydrocyanic acid is formed by a chemical reaction between two substances, a glucoside and an enzyme, contained in the plant. Neither of these substances is poisonous by itself, and under normal conditions they are not in contact with each other. In damaged plants, however, an amount of hydrocyanic acid sufficient to poison animals is sometimes formed by the mixing of the glucoside and the enzyme.

The quantity of potential hydrocyanic acid that can be formed in plants may vary considerably with the stage of growth, the climatic conditions, and the character of the soil. In general, mature cyanogenetic plants contain a smaller percentage of potential acid than young plants. Plants grown on poor soil contain less than those on good soil and fertilization with nitrates markedly increases the percentage of the acid. Sorghums grown in the southern states have not poisoned live stock as much as those grown farther north. The reason for the difference is probably climatic, but little is known about the exact causes of the formation of the acid in this case.

From thorough curing or drying most plants lose the ability to produce acid when eaten by live stock. Some plants, however, retain a considerable proportion of the active acid, even though dried. This applies especially to seeds. Dry, immature flaxseed has caused poisoning. Dry rangoon beans, bitter almonds, peach kernels, and wild cherry bark contain much potential hydrocyanic acid.

Hydrocyanic acid acts very quickly, frequently killing the animals within a few minutes, although sometimes the poisoned animal may live for several hours after the symptoms develop.

Some symptoms observed in hydrocyanic acid poisoning are:

1. The head is turned toward the side when the animal is lying down.

2. The pupils of the eyes are dilated and a watery discharge is given off.

3. The tongue is partially paralyzed and large amounts of saliva run from the mouth.

4. The limbs and ears are cold and the pulse is not perceptible.

5. In the last stages, the limbs are paralyzed and death follows after unconsciousness.

6. In some cases bloating occurs, which is not always relieved through the use of the trocar and canula.

7. Animals have been observed to have become paralyzed within 15 minutes after starting to eat Sudan grass which was damaged.

Six grains of pure acid is considered a fatal dose for the average horse. Cows are somewhat less susceptible. If a plant contains as little as 0.02 per cent potential hydrocyanic acid and, if a horse or cow rapidly consumes it all, 5 pounds of the plant will prove fatal, if no factors enter in to prevent the development of the acid.

According to Prof. Chas. F. Rogers, a biochemist at the University of Minnesota, cyanogenetic plants that are consumed and come into direct contact with the walls of the rumen cause immediate poisoning. The lack of toxic symptoms in some animals eating the same cyanogenetic forage as poisoned animals may be due to the fact that the plants were mixed with other ingesta in the rumen, thereby preventing the poison from coming into contact with the mucous membrane to allow absorption. In the meantime, the poison may have been rendered inert by the digestive processes.

Even when plants contain considerable quantities of potential hydrocyanic acid, they may not poison live stock. Since poisoning depends upon the presence of actual hydrocyanic acid, anything that prevents its development in the stomach lessens or entirely removes the danger of poisoning. Certain feeds, such as alfalfa hay and linseed cake, retard the production of hydrocyanic acid and may prevent poisoning. Glucose markedly represses the rate of formation of the acid. It has been observed

that cattle on a corn ration are less likely to be poisoned when grazing on dangerous sorghums than cattle that are not fed corn. It is therefore a wise precaution to give animals a starchy feed, such as corn, when feeding Sudan grass.

An alkaline condition of the rumen may diminish the possibility of poisoning. The greatest danger from poisoning is usually that of wilted plants, such as cherry leaves from a broken branch of a tree, Sudan grass or sorghums.

The treatment for hydrocyanic acid poisoning has been very successful. The combinations include potassium permanganate, methylene blue, sodium thiosulfate, sodium nitrite and glucose. Most of the pharmaceutical houses have preparations, either in the tablet form or in solution.

One treatment is composed of 30 per cent sodium thiosulfate and 10 per cent sodium nitrite; another is composed of 1 per cent methylene blue, 25 per cent dextrose and 10 per cent sodium thiosulfate; another uses 30 per cent sodium thiosulfate and 2 per cent of sodium nitrite. These solutions must be given intravenously.

On animals not prostrated, gastric lavage may be used to remove much of the poison forage. Following removal, quantities of warm water containing several ounces of sodium thiosulfate pumped into the rumen may help to overcome the potential poison in the rumen.

Adrenal Lesions in Grass Tetany

In a study of two cases of grass tetany, Montaux (*Rec. de Méd. Vét.*, August, 1939) found marked lesions of the adrenal glands. In one case the right adrenal was the size of a large apple. In fact, the gland was replaced by encapsulated tumorous tissue of encephaloid consistency strewn with hemorrhagic spots and calcareous grains. In the other case the adrenals were polycystic, with a tendency toward necrosis.

Important neoplastic lesions of the kidneys and abdominal lymph glands also were disclosed. Lesioned liver is well known to exist in grass tetany and similar affections (milk fever), and this factor seems to play an important rôle in their appearance and evolution. It would, however, be premature to hold that grass tetany and milk fever are manifestations of adrenal insufficiency, although all agree that these diseases are autointoxications occasioned by a brisk change in the physiological state: parturition, railway voyage, change of alimentation. These lesions of the adrenals merely support the logical antitoxic theory, the theory of physiological upset, in which the organs charged with checking, fixing, transforming and eliminating the toxic molecules are not functioning in regular order. In other words, the lesioned organs found post mortem preëxist and are therefore the cause, not the effect, of the disease.

Chemotherapy in Bang's Disease

A number of capable authorities have expressed the opinion that the greatest hope in the handling of Bang's disease lies in chemotherapy, that is to say, in specific microbicide medication. The belief is based to a considerable extent upon reported benefits derived from the use of certain dyes in undulant fever of man in the Mediterranean littoral, where Malta fever is a common interchangeable malady among the human and ruminant populations.

Although it is easy to be deceived in the use of drugs or any remedy in such a self-limiting disease as undulant fever, the benefits derived from certain acridine dyes seem incontestable, according to the constant reports one reads in the medical literature of France. The result of drugs seems to be more promising in human brucellosis of bovine origin, these reports contend, because of being more benign than the melitensis infection contracted from goats.

The fact that sulfanilamide has a favorable action in human brucellosis of caprine origin is encouraging, since it may forecast the coming of a chemical cure of this ubiquitous and troublesome affection of cattle, a cure that would aid, and in no measure obstruct, the eradication program.

Effect of Anti-Encephalomyelitis Serum on Subsequent Production of Active Immunity with Encephalomyelitis Vaccine

By WILLIAM S. GOCHENOUR. JR.,* V.M.D.
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DURING an outbreak of equine encephalomyelitis immediate danger of exposure of animals not preseasonally immunized with vaccine may necessitate the use of encephalomyelitis antiserum to afford immediate protection. The problem arises as to the method to be employed to convert later this passive immunity to an active immunity through the use of vaccine. In previous years lack of experimental data relative to the method of choice led to a diversity of procedures; many administering the first dose of vaccine simultaneously with the administration of the serum, some allowing a week to elapse between the administration of the serum and the first dose of vaccine, and others waiting for two weeks after serum administration before instituting vaccine treatment. In other instances the appearance of danger within a few days after the first injection of vaccine necessitated the use of antiserum, and this was subsequently followed by the second injection of vaccine.

To gain information as to the effect of the antiserum on active immunity production by these methods, a series of experiments was conducted, employing guinea pigs as test animals, since this species is especially suited for encephalomyelitis immunity investigations.

Results of experiments in equine encephalomyelitis vaccination.

GROUP NO.	TREATMENT	NO. IN GROUP	SURVIVALS	
			NO. ANIMALS	PERCENTAGE
I	Two injections of vaccine seven days apart; exposure ten days after second injection of vaccine	10	10	100
II	First injection of vaccine followed at five days by serum injection, followed at ten days by second injection of vaccine; exposure ten days after second injection of vaccine	5	4	80
III	Serum injection simultaneous with first injection of vaccine; second injection of vaccine seven days after first; exposure ten days after second injection of vaccine	7	0	0
IV	Serum injection seven days preceding first injection of vaccine; second injection of vaccine seven days after first; exposure ten days after second injection of vaccine	8	1	12.5
V	Serum injection fourteen days preceding first injection of vaccine; second injection of vaccine seven days after first; exposure ten days after second injection of vaccine	11	9	81.8
VI	Controls; no serum or vaccine administered	4	0	0

Guinea pig weights—450 to 500 gm.
Vaccine injections—0.5 cc. subcutaneously.
Serum injections—1.0 cc. subcutaneously.
Virus injections—0.1 cc. of a 1:500 dilution of infected guinea pig brain intracranially.

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Six groups of guinea pigs were used. Group I, consisting of ten animals, was given two injections of vaccine alone seven days apart and exposed to an intracranial inoculation of virus ten days after the second injection of vaccine. Group II, consisting of five animals, was given one injection of vaccine followed at five days by an injection of serum, this in turn followed at seven days by the second injection of vaccine, and the animals were exposed on the tenth day following the second vaccine injection.

Group III was given two injections of vaccine seven days apart and one injection of serum simultaneously with the first dose of vaccine. The experimental animals were exposed on the tenth day following the second injection of vaccine.

Group IV was given two injections of vaccine seven days apart and one injection of serum seven days preceding the first injection of vaccine. The guinea pigs were exposed on the tenth day following the second injection of vaccine.

Group V was given two injections of vaccine seven days apart and one injection of serum 14 days preceding the first injection of vaccine. This group also was exposed on the tenth day following the second injection of vaccine.

Group VI comprised controls given no serum or vaccine.

The results of this series of experiments are shown in the accompanying table.

One hundred percent protection was afforded all of the animals in group I, each of which received two doses of vaccine alone.

In group II there was evidence of some neutralization of the antigen of the second dose of vaccine by the serum administered, and the degree of active immunity produced was not as high as that in group I.

In group III complete neutralization of the vaccine by the serum occurred and no active immunity was developed.

In group IV there was marked neutralization of the vaccine, with the development of very little active immunity.

In group V, however, the neutralizing capacity of the serum had largely been dissipated, and a good degree of active immunity had been produced.

From these experiments it appears inadvisable to administer the first injection of encephalomyelitis vaccine simultaneously with or earlier than two weeks subsequent to the administration of encephalomyelitis antiserum when attempting to extend the immunity conferred by the serum with active immunity by the use of vaccine.

Origin of the Ossification of the Lateral Cartilage of the Hoof

It is mentioned in the literature that the ossification of the lateral cartilage of the hoof starts at the angle of the coffin bone, but no particular place of origin is pointed out. Müller's investigation disclosed that the ossification starts always at a certain definite site of the angle of the coffin bone. Insular ossification was found in ten of the 41 hoofs examined.

The ossification, starting at the angle of the coffin bone, as well as the one starting as an island, is brought about by conditions differing from the normal enchondral ossification. Responsible for this condition is the direct transformation of the basal, fibrous, cartilaginous substance into osseous tissue. The starting points for the insular ossification are cavities which are often found in the cartilage of the hoof. They contain undifferentiated connective tissue, fibrils, and vessels. The process of ossification of the lateral cartilage of the hoof may terminate when the connective tissue changes to fatty tissue, and when it loses its ability to form osteoblasts. *(Über den beginn der Hufknorpelverknöcherung (The Origin of the Ossification of the Lateral Cartilage of the Hoof), by E. Müller. Tierärztl. Rundschau, xlv 1939, p. 90.)*

Pruritus vulvae and pruritus ani, two troublesome ailments of the human being, are said to yield to the administration of nicotinic acid.

LEGAL OPINIONS

By JOSEPH M. KOTZ

Attorney for the Association

This brief inclines to cover in a general way certain juridic ramifications of veterinary practice; to point out some of the legal pitfalls of the uninformed practitioner; to consider the veterinarian's social responsibility as conceived by the courts.

THE CANONS and ethics of veterinary societies are much more exacting than those deliberated by law. The statutes in most of the states prescribe the qualifications and requirements necessary for one to be admitted as a practicing veterinarian. To illustrate: If one is to qualify for admission to the profession, he must be of good character and morals. On the basis of this broad ruling, the committees of these societies can, if they feel that the transgressions warrant, recommend to the state board of veterinary registration that the doctor's license to practice be revoked.

To the extent that the state governments exercise certain privileges and determine in a large measure the conditions under which medical men must practice, the veterinarian is a public servant. By virtue of his work, he has certain definite social responsibilities. He contacts with the law in many ways, not only through actions for his own alleged malpractice [discussed in the February, 1939, issue of the JOURNAL] but also for any negligent acts of his employés.

Categorically speaking, there is no law that requires a veterinarian to treat an animal if he does not wish to do so. Of course, he may be criticized for refusing to administer aid and thereby lose considerable prestige and business. But, once a veterinarian undertakes to treat an animal, he must continue or else allow the owner a reasonable opportunity to secure another doctor. Failure in this respect makes him liable to the owner. Naturally, if the owner requests him to abandon the case, he is relieved from further legal liability.

It has been ruled that when a veterinarian accepts a case, he impliedly represents himself to be equal to the undertaking and to possess the degree of skill and learning that characterizes the profession of his locality. It becomes his responsibility to use his best judgment and reasonable care and diligence to accomplish the task he has accepted. Further, he is personally liable for the conduct and all of the acts of his assistants. If such assistants are furnished to him at a hospital where he is merely performing an operation, however, he does not become liable for their actions.

The question of what constitutes a reasonable fee for services rendered often enters the legal domain. Basing the fee on the financial ability of the owner to pay is unlawful. The courts have pointed out that in deciding what is a fair and reasonable charge, the law must consider the ability and reputation of the doctor, the demand for his services, the importance of the case involved, the time spent, the prevailing charges among other veterinarians in the given locality, and the results secured. None of these in itself determines precisely the proper fee for services.

Diplomatic Immunity Claimed for Cocker Spaniel

Mrs. Heinz Thorner, wife of an attaché of the German consul general, has claimed diplomatic immunity for her Cocker Spaniel, Purzel. She allowed the dog to run without a muzzle or leash around Forest Hills, Long Island, and was summoned to court, where she claimed diplomatic immunity. The magistrate reserved his decision.

CURRENT LITERATURE

ABSTRACTS

Autohemotherapy in Horses

Treatment by means of administering the patient's own blood, previously exposed to ultraviolet rays, has been tried upon 43 horses suffering from the following ailments: chronic phlegmon, acute and chronic arthritis, chronic bronchial catarrh, emphysema of the lungs, cerebral paralysis, spinal paralysis, and disorder of His' bundle. The following observations have been made: Autohemotherapy always produces a definite general reaction of the body. The curative effect is greater than with untreated blood. There is a distinctly anodyne action. Treatment is tolerated well at all times. Complications do not occur. Certain cases which fail to respond to the usual line of treatment have responded with healing or at least improvement to the autohemotherapy. *(Die Behandlung mit ultraviolet bestrahltem Eigenblut beim Pferde (Autohemotherapy in horses), by H. Steffan. Zeitschr. f. Veterinark., li 1939, pp. 147-169; 195-217.)*

Intravenous Injections of Carbon (Granulotherapy)

Among the indirect antiseptic methods intended to excite the different defenses of the body, the use of proteins and colloids is the most important. Granulotherapy is a new modification of colloidotherapy. For this purpose charcoal, in a concentration of 2 per cent in distilled water, normal salt solution or glucose, is employed.

To be utilitarian as an intravenous injection, the suspension should conform to certain physical conditions, the most important of which is the dimensions of the grains. The ideal suspension is one containing only round grains of 5μ to 15μ in diameter. While the perfect product has not been realized, the animal charcoal of the market is practically inoffensive, when employed in doses that do not block the reticulo-endothelial system.

For dogs the posology is as follows: Two to three injections of 2 to 3 cc. every day or two. For horses the treatment is somewhat dangerous because of their sensitivity to "shock" substances. The same danger does not exist in cattle, however. It is recommended that a large syringe lubricated with paraffin oil be used, to prevent gripping of the piston. The mode of action is complex and controversial.

Hyperleukocytosis and elective fixation of the carbon on the reticulo-endothelial system are, according to Lumière, two established facts. But, it must not be concluded here that the excitation of the defenses is the only action. There are also chemophysical actions to consider, such as augmentation of the alkaline reserve, diminution of the protidemia, raising of the serum-globulin quotient, chloruemia and glycemia. The humoral action is effected through the intermediary of the adrenalino-sympathetic system because the glycemia does not appear if that system is experimentally paralyzed.

There is nothing astonishing about the action of carbon when one considers the action of protein and colloid on the nervo-vegetative system. In regard to the phenomena of defense, they can be manifested as well through the intermediary of the adrenalino-sympathetic system as through that of the reticulo-endothelial. Moreover, according to Brocquet and Saint-Jacques, it also plays the rôle of an absorbent in the presence of toxins.

Whatever may be the mode of action of intravenous injections of carbon suspensions, they are therapeutically harmless and efficacious, although not universally so.

For example, they are not effective during the septicemic period of canine distemper. But, in postpartum infections they render an important service.

Although this observation was made only upon dogs, the carbon-suspension treatment has been employed for several years on large animals at the veterinary school of Toulouse. The doses recommended by Cuillé and Chelle are, for cattle, 20 cc. of the 2 per cent suspension. The injections can be safely repeated every day. They have been employed not only for postpartum troubles but also for grave diseases of the respiratory organs. *(F. Liegeois and P. Terache. Annales de Médecine Vétérinaire, October, 1937. Abstract in Revue de Médecine Vétérinaire, xci, June 1939, pp. 349-350.)*

Leukosis in Domestic Animals

Leukosis is a tumor-like granulation of a blood-forming tissue. Depending on the degree of maturation of the cells, one may classify the leukosis as myelosis, lymphadenosis, lymphosarcomatosis, and primary cell leukosis. In domestic animals the aleukemic form predominates. Cattle are most commonly subject to leukosis. In mountainous countries the disease is rare. The infection theory possesses little probability etiologically. Nevertheless, a living or non-living contagion can not be excluded entirely. The lesions of chronic protein poisoning exhibit so much similarity to bovine leukosis that an etiological connection seems plausible. The disease agent is present in the blood serum and in the lymph glands. The rôle played by other factors, such as management, feeding, individual disposition, heredity, etc., in the causation of leukosis is not known.

As a rule, bovine leukosis appears in cattle aged 5 to 8 years but may also be observed in calves. Females seem to become affected more often than males. Usually, the course of the disease is chronic. Depending upon the blood picture, bovine leukosis may occur in a leukemic, subleukemic, or aleukemic form. The question of hereditary transmission can not be answered yet, but placentary transmission is proved. Bovine leukosis possesses much similarity to avian leukosis. *(Leukosen der Haustiere (Leukosis in Domestic Animals), by O. Seifried. Tierärztl. Rundschau, xlv 1939, p. 26.)*

The Consequences of Bovine Brucellosis

The loss in milk following an abortion from Bang's bacillus infection is moderate but continues to an even more appreciable degree during the next year. The continuation of diminished lactation is imputable to the marked systemic weakness that establishes itself in infected cows. The weakness is due to the fact that cows are not given the time to overcome the malady.

A new gestation immediately following abortion only gives the infectious agent another favorable field for growth and does not give the cow the chance to excrete the organisms. An animal exposed to an intermittent infection through the years does not attain its productive value to the same extent as one allowed to triumph over its ailment.

A cow after aborting that is not exploited for milk production, that is well fed and managed, surmounts the disease and recovers its health, while one immediately bred remains systemically depressed. Six months should elapse before an aborting cow is rebred. Inasmuch as the disease is one of the genital organs, each abortion requires the advice of the veterinarian on the sanitary measures to be employed in preserving the health of the animal, for at the present time there is neither vaccine nor drug that gives satisfactory results. *(Heck. Deutsche tierarztliche Wochenschrift, xlvi, April 9, 1938, pp. 228-232.)*

Causes of Abortion Other Than Brucellosis

Gillman (*The Cornell Veterinarian*, April, 1939) names and briefly describes various causes of abortion in cattle other than Bang's disease. In view of the fact that owners of herds known to be free of brucellosis are always perturbed when abortions

occasionally occur, the other factors responsible for abortion should be kept in mind. The causes of bovine abortions listed by this author are:

1. Trichomonads
2. Spirilla (Smith)
3. *Bacillus pyogenes*
4. Streptococci
5. *Bacterium paratyphosus*
6. Fungi and moulds
7. Tuberculosis
8. Miscellaneous bacteria
9. Vesicular venereal disease
10. Smut, ergot, rust
11. Nutrition
12. Endocrines
13. Injuries, shipping
14. Miscellaneous causes
15. Unknown causes

Since brucellosis and trichomoniasis can be readily detected by capable clinicians—and these are by far the more common causes of premature births in cows—the sporadic visitations named above need not materially cloud the issue where the principal causes have been eliminated.

At Columbia University are 2,000 healthy rats. They are the current survivors of 45 generations of rat life, representing 900 years of human life. Some of the rat families have lived on one diet—wheat, milk, table salt and distilled water—through the whole 45 generations.—*Edwin Miller in The Forum.*

BOOK NOTICES

Eastern Iowa's Souvenir Book

The Eastern Iowa Veterinary Association wound up its first 25 years by writing a book—a history of its birth, growth and achievements which, as the title page says, was "compiled, composed and edited by amateurs between professional calls." Whether that pronouncement was intended to parry the literary critics or not, it tells in one clause just why "Eastern Iowa" in the veterinary circle has become synonymous with successful professional work, for the medical man who does not find it possible to take time out to aid in keeping up the fences of his profession doesn't amount to much.

A look into Eastern Iowa even before this association was formed would reveal a group of animal doctors who were fired with the determination to be useful not only to their region but also to the occupation they had chosen. The formation of a local society that would attract nationwide attention for resourcefulness was therefore a natural sequence.

"25th Anniversary Souvenir Book, 1913—1938" of the Eastern Iowa Veterinary Association is 156 pages, 10½" x 7½" of important data written in chronological order by various members in key positions who set down in print what may justly be regarded as a true history of veterinary development in the Corn Belt for the period covered. Veterinary historians of the future will find this book a precious account of significant events as well as a complete directory of the past and present veterinarians who have labored professionally on that terrain.

Portraits, photographs of groups, snapshots from the summer clinics, all of which are woven into the Association's affairs, take up 21 pages which, in themselves, tell a great story without printed words—a story of initiative, foresight, achievement—and the reproduction of press clippings that fill local newspapers month after month with publicity of the useful sort has taught a lesson that even the national association has been prompted to imitate.

This souvenir book is interesting, fascinating, utilitarian, and on every count a credit to its editors and sponsors. *(25th Anniversary Souvenir Book, 1913-1938, Eastern Iowa Veterinary Association. Dr. Iva Dunn, business manager, Aikins, Iowa, 1938. Price, $3.00.)*

Canine Pediatrics

A brochure of 78 pages on the subject of nutrition in puppies, illustrated with numerous graphs and pictures, consists of well chosen articles recently printed in *Veterinary Medicine*. Its text is based upon experimental work carried out at the Borden research laboratories by Supplee and LeWare for the purpose of developing from cows an equivalent of bitch's milk that can be employed successfully in the practice of canine medicine.

This little booklet condenses the sum of biochemical knowledge which has made infant nutrition a specialized science, a science which overcomes the analytical differences between the milks of the different mammalian species and which also audaciously aims to, and does, improve upon (balances) the milk of a nursing mother for her own offspring.

Canine Pediatrics, on the one hand, is highly educational and on the other it is a useful reference for the moment when artificial feeding of a litter of pups has to be resorted to from any cause. It is based upon modern concepts of digestive and nutritional physiology and the intricate chemistry of the most precious of all foods —milk. The use of the word "pediatrics" in animal medicine is not as much of a strain on usage as it may seem at a glance. *(Canine Pediatrics, by D. M. Campbell, G. C. Supplee and J. H. LeWare. 78 pages, illustrated. Veterinary Magazine Corporation, Chicago, 1939. Price, $1.00.)*

A Guide to Veterinary Parasitology and Entomology

From professors of the University of Liverpool comes an excellent manual on parasitology and entomology, for veterinary students and practitioners, which condenses a text on two broad subjects into 176 pages of illustrated reading material. The condensation into a "guide" is well executed and the ground covered is quite extensive. As an introduction to these subjects and as a plan of study, this volume leaves little to be desired. The illustrations, made from drawings, are far above the average for clarity and for the features they are intended to portray. In this respect, the coccidia and blood protozoa are particularly deserving of praise.

Twelve full-page diagrams illustrate the life histories of that many cosmopolitan worm parasites of animals. These diagrams are extremely well d e l i n e a t e d. Though the descriptions are necessarily brief and the number of described parasites few, the actual amount of information contained is indeed surprising. The book is pleasant reading throughout.

As a book on entomology, there is disappointment on account of the few pestiferous insects included. Obviously, the authors did not intend to dwell upon the potentiality of the phylum Arthropoda in insect-ridden countries. *(A Guide to Veterinary Parasitology and Entomology for Veterinary Students and Practitioners, by T. Southwell, D.Sc., Ph.D., etc., and A. Kirshner, M.B., Ch.B., etc. Second edition. 123 illustrations, 12 diagrams, 176 pages. R. K. Lewis & Company, Ltd., London.)*

The Springer Spaniel

"The Springer Spaniel," the latest addition to the group of dog books published by the Judy Publishing Company, is a volume of 160 pages divided into 38 chapters. The first 16 chapters are devoted mainly to the title, and the other chapters contain condensed general information applicable to any dog and any breed.

The history of this aristocratic gun dog is traced back to medieval times and pedigrees as far back as the first decade of the 19th century. The blood of Ch. Velox Powder (1912) courses through the veins of many a Springer of the present, notwithstanding that the breed was benched for the first time under that name in 1903. Under the name of English Spaniel, the Springer was the common village dog of New England before the Civil War.

Bench show standards of the breed are graphically portrayed by photographic reproductions and drawings.

The last 22 chapters contain condensed general information on dog breeding and management in a fashion that prompts a reviewer to laud the author for his sound advice. He apologizes for the brevity of the single page on "Diseases of the Dog" in saying that "veterinary services are reasonably available in all places" with few exceptions, and in reminding his readers that "still the best of all doctors is common sense." These two remarks qualify the author for honorary membership in veterinary societies.

The chapter on laws about dogs contains the definite information in this connection that everyone who harbors a dog should know, and in "The Springer Spaniel" one finds useful information on benching, shipping, training, kennel management, sales, advertising, feeding, etc., all boiled down into short chapters of exceedingly pleasant reading and informative material. The veterinarian should have this book in his library and recommend it to his clients regardless of the breed they fancy. *(The Springer Spaniel, by Maxwell Riddle, breeder, judge, journalist. 160 pages, illustrated. The Judy Publishing Company, Chicago, 1939. Price, $2.00.)*

Epidemic Encephalitis

The third report of the Matheson Commission, which was formed to study epidemic encephalitis (encephalitis lethargica), is published in the form of a book of 494 pages, 298 of which comprise an extensive bibliography gathered from the principal countries of the world. The Commission's first report was published in 1927 and the second in 1932.

One of the theories of etiology unfolded is that the disease may be due to a neurotropic form of herpes virus, a theory signalized by Levaditi. Much work has therefore been done in developing a herpes antigen for immunizing purposes. This phase of the research is described at length in the opening chapter.

The Commission has deemed it justifiable to consider in detail every therapeutic agent that has been claimed to have merit. Nearly 40 chemical, galenical and biological remedies are analyzed for their curative or palliative properties. Among these are atropine, scopolamine, stramonium, salicylates, iodine, quinine, pilocarpine, and arsenic compounds. The biological products described are immune horse and rabbit, formalized herpes virus ("F" vaccine), convalescent serum, cobra venom, and endocrine derivatives. One of the remedies dwelt upon at length is the Bulgarian treatment, consisting of a decoction of belladonna root and calamus made by a special process with white wine. For modifying the rigidity and tremors, belladonna grown in Bulgaria and Italy appears to have special value.

There is no evidence that the infection is transmitted except by human contact, contrary to the virus of equine encephalomyelitis, which may be transmitted by several varieties of mosquitos.

The Massachusetts outbreak (1938) affecting children with the eastern type of equine encephalomyelitis in acute form is described, but the equine infection is not listed in the chapter on "Various Allied Diseases."

Although the Commission does not attempt to predict the future behavior of the disease, the fact remains that the incidence has been declining since the "waves" of 1920 and 1926.

The chapter on epidemiology contains a wealth of statistical records from foreign and domestic sources. *(Epidemic Encephalitis, edited by the Matheson Commission for the Study of Epidemic Encephalitis. 494 pages. Columbia University Press, New York City, 1939. Price, $3.00.)*

Veterinarians Are Grateful, Doctor

From widely separated parts of the country, members of the Association reminded us of a compliment paid to the veterinary profession by a New York physician over NBC, Sunday, July 9. At the spelling bee between physicians and nurses, the conductor, in apologizing to one of the physicians for choosing words that were strictly veterinary, remarked, "Doctor, you're not a veterinarian, are you?" Whereupon, the doctor replied, "No, but if I were a good one, I would be proud of it."

The meaning of this is that the fine compliments paid to the veterinary profession in radio programs are one of the signs of the times brought about by our good behavior.

From remote antiquity to the present day, most of the knowledge on horse diseases was handed down to the civilian population by the military veterinary service.

THE NEWS

*Veterinary Contributions to Human Health**

A FRACTURE SPLINT, originally designed and used to set fractured bones in dogs, has recently been used so successfully on humans with fractures that they can walk without the aid of crutches in as short a time as ten days. Previously, with older methods, it was necessary for such patients to remain in bed, often in very uncomfortable positions, for as long as two months.

This is but one recent example of how man has benefited from research work on the illnesses and diseases of animals. The present health, vigor and long life expectancy of the nation is a result, to a large extent, of the prevention and cure of infectious disease in our cattle, swine, sheep, poultry, horses and dogs.

The country's 13,000 veterinarians have been in the forefront of this battle against animal and human disease. For the past 76 years—during which the American Veterinary Medical Association has been in existence—veterinarians have striven unceasingly to solve problems of animal health, many of which have a direct bearing upon human health.

MAN IS SUBJECT TO 70 ANIMAL DISEASES

The yearly convention of the A.V.M.A. is an annual milepost in the never-ending struggle to relieve mankind from the approximately 70 diseases and parasites which are communicable from animals to man. The country's leading scientists, bacteriologists, animal surgeons and teachers of veterinary medicine report to the convention on the progress of their work during the year.

The convention this year—to be held from August 28 through September 1 in Memphis, Tenn.—promises to be more fruitful than any of the 75 meetings previously held. Many significant developments are to be revealed.

In the past, announcements of the conquest of the bovine type of tuberculosis, of foot-and-mouth disease, of anthrax, of malaria, and of other dangerous diseases from which mankind has suffered, have been widely heralded.

Today, we may look forward hopefully to the eventual complete conquest of rabies, Bang's disease, mastitis, equine encephalomyelitis and other animal diseases which have a close bearing on human health.

LIVE STOCK IS NATION'S LARGEST INDUSTRY

Since the beginning of time, man has depended upon the meat of animals for much of his food. Today, the livestock and meat industries are declared to be the nation's largest, with live stock accounting for one-fourth of the total United States income from agriculture.

This could not be true, however, if the United States were not the safest place in the world to produce live stock. For, without the veterinary supervision now in operation, plagues caused by disease would quickly wipe out millions of meat-producing animals and result in the deadly famines which formerly occurred periodically in all the civilized nations of the world.

Such plagues would not only diminish the country's meat and milk supply, but every farm crop would suffer because horses would become infected, and horses are vitally important to millions of farmers. In our orchards, our groves, our berry patches and on our farms, there are no less than 13 million horses and mules at work.

RUSSIAN FAMINE DUE TO VETERINARY CURTAILMENT

Russia, following the 1917 revolution, suffered the most devastating animal plague that has occurred in any nation in more than a century. This brought about widespread pestilence, causing millions of people to die either of outright starvation or of diseases directly or indirectly resulting from lack of food. The reason for this deplorable situation was that the revolutionists had replaced the qualified veterinarians with laymen selected largely from the army. The lesson was learned. Today, Russia has one of the most elaborate veterinary services in the world.

In the United States, the veterinarians who work in the Bureau of Animal Industry of the U. S. Department of Agriculture do yeoman

*Prepared by President Bergman in correlation with an extensive program of publicity, to be continuous throughout the 76th annual meeting of the A.V.M.A., to focus the attention of the public on the significance of veterinary service, and to attract attention to the activities of the convention. Copies of this report are now in the possession of every recognized newspaper service in North America.

service in eradicating diseases from the animal population. Secretary of Agriculture Wallace recently declared, "The federal meat-inspection service has attained a plane of efficiency and protection unexcelled by that of any other nation."

As a result, most families today buy their food without a thought as to its purity and safety. They take that for granted. They buy their meat intent only upon getting a choice cut at the right price. They buy milk, cream, butter, cheese, eggs and poultry also with carefree assurance.

Hunchbacks Decrease as Tuberculosis Wanes

Very few hunchbacked persons are seen nowadays who are less than 20 years old. Leg and joint deformities are fewer. For, the bovine type of tuberculosis—which causes these dread afflictions—is under control.

The past 20 years, the U. S. bureau of animal industry and coöperating livestock and sanitary officials and practicing veterinarians in all states have been waging a war on bovine tuberculosis. These allied veterinary forces have been testing from one to two million cattle a month and have already removed and slaughtered more than 3½ million tuberculous animals. Twenty years ago, the average infection was about five animals in 100. Today, scarcely one in 200 shows the disease.

The healthy cow has been called the "foster mother" of the human race. Milk is not only our most nearly perfect food but one of our most vital foods. And yet, it may under certain conditions be a dangerous food.

The heart of the problem of a safe milk supply is a healthy cow. Cows are subject to certain diseases, such as tuberculosis, which may make their milk products unsafe for consumption. Veterinary inspectors constitute the first line of defense of the nation's pure milk supply. They are leading the way in the fight against Bang's disease and mastitis. They are assuring a dependable supply of nature's "most perfect food."

Panama Canal Made Possible By Veterinary Discovery

The Panama Canal would not have been constructed if a group of veterinarians, who were fighting Texas fever in cattle, had not discovered that it is an insect-borne disease. Up to that time, the Panama Canal could not be built because of yellow fever and malaria. The scientists working on yellow fever were given their cue by the discovery of the cause of Texas fever.

Following this line of research, these scientists soon found that yellow fever is transmitted to man by a mosquito. They then sought and found methods of eliminating the mosquito. With this accomplished, yellow fever was controlled and the Panama Canal was completed.

Also as a result of this discovery, an estimated three millions of our population, principally in the South, who are now well, would be sick continuously with malaria.

Hookworm Treatment Followed Dog Discovery

There are many other contributions which veterinary research has made to our knowledge of the diseases of humans. For example, the treatment of hookworm in man, now used to the extent of millions of doses annually, was discovered by veterinarians experimenting in the treatment of the same parasite in dogs.

Foot-and-mouth disease not long ago was one of the most virulent diseases transmitted from animals to man. The discovery that the virus of this disease reached this country in smallpox vaccine led to its eradication.

Anthrax is a disease as old as Greek civilization which has been brought under control. It is spread by contaminated hides, wool from affected sheep, and by other means. It is another of the animal scourges of mankind which veterinary science has helped to curtail.

There have been other broad human results from veterinary research. The study of malnutrition and its treatment in live stock and fowl has proved very helpful in dealing with metabolic diseases in man. Also, the great progress in the knowledge of animal feeding has created new possibilities for better nutrition in man.

Veterinarians Recognize Their Responsibility

It may be seen, therefore, that the nation's health and vigor are dependent to a large extent upon the disease-prevention work of its veterinarians. The veterinary profession recognizes its present and future responsibility in contributing to public health and is proud of the service it has been able to perform in the past.

Finds Nicotinic Acid Will Cure X-Ray Ill

A Toronto, Ont., physician, J. Wallace Graham, has discovered that the "radiation sickness" which often accompanies x-ray treatment can be combated effectively with nicotinic acid. A recent issue of the *Journal of the American Medical Association* carried the report of this finding, wherein it was pointed out that the nicotinic acid therapy gave definitely satisfactory results in three-fourths of the cases studied.

The Veterinarians of the Month

Chief Mohler Honored

Advance information from the Special Committee on Twelfth International Congress Prize names John Raymond Mohler, chief of the Bureau of Animal Industry, United States Department of Agriculture, as the member of the Association who is entitled to receive the International Congress Prize for 1939. The prize will be awarded in behalf of the A.V.M.A. by H. W. Jakeman, chairman of the Executive Board, at the opening session of the Memphis meeting.

John R. Mohler

[The precocious announcement of this event of the meeting is not a violation of the confidence in the editorial room, since the present issue will not have been mailed at the time the prize is awarded.]

G. P. Statter Rides into the News; Wins Publicity for Veterinary Profession

When Glenn H. Campbell, addressing the recent nutrition conference at Decatur, Ind., urged veterinarians to seek more prominent rôles in civic activities, he certainly could not have had in mind one George Pierrpont Statter of Sioux City, Iowa. For, it is Dr. Statter, practitioner, who has synonymized his professional skill with a civic entity—the Abu-Bekr World Famous White Horse Mounted Patrol—that yearly wins the attention and plaudits of many thousands. A brief history of the Patrol will identify Dr. Statter's part in this unique organization.

G. P. Statter

The Abu-Bekr White Horse Mounted Patrol was first conceived in 1920 by a group of livestock men, members of the Abu-Bekr Shrine Temple, who visualized a mounted patrol, stellar in its kind, composed of dashing white Arabian mounts that would one day occupy a spot of singular prominence, giving to the Abu-Bekr Shrine Temple and to Sioux City a possession unequaled anywhere in the world.

A determined and exhaustive search revealed that horses of desirable qualifications were absolutely unobtainable anywhere in the world. In the face of this, these men utilized the material at hand, filling in with horses of almost every color and type. Not to be deterred from their original purpose, however, and realizing their inability to purchase horses of the desirable qualifications, they resolved to raise their own. To this end, they procured from the far South a pure white, dashing Arabian stallion, pink skinned, proud of bearing and directly descended from the far-famed steeds of the Arabian desert. This magnificent horse was placed at the head of the Patrol and named for the Shrine Temple—"Abu Bekr."

Today, although slightly aged, this proud sire maintains his place in the troop and, ridden by First Lieutenant Statter, takes a leading part in every performance. The majority of the Patrol's proud beauties are sired by this horse. Through the expenditure of thousands of dollars, the Abu-Bekr group has succeeded in acquiring all white mounts, uniform in quality, size and conformation. The Patrol on parade is a truly impressive sight and one received with high acclaim by lovers of horseflesh everywhere.

Aside of parades in the interests of the shrine, the troop has made appearances on many other occasions. Chief among these are horse shows, rodeos, county fairs, Fourth of July celebrations and corn-husking matches. In fact, almost anywhere that a crowd assembles, the Patrol has performed, delighting audiences with its fancy drills and intricate maneuvers.

John R. Mohler, chairman of the committee in charge of the A.V.M.A. New York World's Fair exhibit, announces the receipt of a $50.00 contribution to the fund that is helping to finance the venture, from the Missouri State Veterinary Medical Association. This amount added to previous subscriptions makes a grand total of $3,213.20.

Continuation schools on poultry diseases for veterinarians are conducted by E. S. Weisner (Mich. '37) under the sponsorship of the Michigan State College.

U. S. GOVERNMENT

Regional Research Poultry Laboratory Dedicated

Following a busy fortnight in connection with the Seventh World's Poultry Congress, John R. Mohler, chief of the U. S. bureau of animal industry, in company with prominent foreign delegates proceeded to East Lansing, Mich., to attend the dedication of the Regional Research Poultry Laboratory in that city. In his dedicatory address, Chief Mohler said in part:

> I feel that the federal government and the 25 states coöperating with the Regional Research Poultry Laboratory have been fortunate in obtaining the services of Dr. J. Holmes Martin as director. . . Through public addresses and in the press, Dr. Martin has clearly stated the lines of research being followed in attacking basic problems. * * *
>
> The staff here at East Lansing will, of course, have the benefit of counsel by specialists of the Department of Agriculture and of the coöperating experiment stations extending from as far west as North and South Dakota, Nebraska and Oklahoma to the Atlantic Seaboard.
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> * * *
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> In conclusion, let me emphasize that this institution has progressed beyond its infancy and is now on a vigorous working basis. Its activities are well coördinated and directed toward a definite goal. However, the institution is still young and has yet to develop its mature individuality. I invite your coöperation, therefore, toward molding it for a great career of leadership and usefulness. To this end I now dedicate this Regional Research Poultry Laboratory.

The ceremonies were attended by 250, representing many states and countries. The occasion was a fitting place and moment to announce that the Congress which had just closed at Cleveland had attracted an unprecedented patronage of 800,000.

"How Dangerous Are Wood Ticks?"*

Under this inquiring headline the consumers' counsel division of the AAA offers answers to several of the important questions concerning wood ticks.

There were only about 500 cases of Rocky Mountain spotted fever—the disease caused by the wood tick—in the entire country last year, and but one-fifth of these terminated fatally. By taking reasonable precautions the average person has relatively little chance of becoming infected, since only about one of every 500 or 600 ticks carries the disease. In general, they attach themselves to dogs* or other animals rather than to humans.

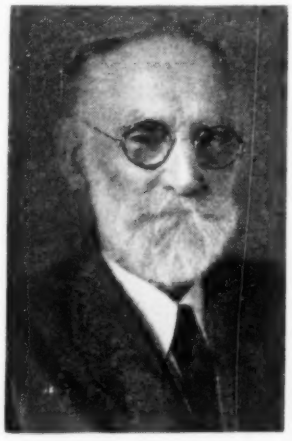
An infected tick must remain on the body for about six to eight hours in order to transmit spotted fever, and the disease itself requires four to twelve days to develop. The first symptoms are commonly a chill, fever, and a very severe headache.

Should you live in a tick-infested area, the best precaution is to look yourself over thoroughly, particularly your hair, twice a day in order to determine if any ticks have become attached. An occasional glance at your clothing is also recommended. If a tick is present on your body, do this: Pluck it off with tweezers, or your fingers, being careful not to crush it. Destroy the tick by dropping it in kerosene or boiling water. Next, put iodine on the exact spot where the tick was launched. Be sure to dip your fingers or the tweezers in alcohol, or wash thoroughly with soap and water, after the tick has been removed.

*Consumer Notes, ii (August 7, 1939), 15, p. 1. Issued by the consumers' counsel division of the AAA, Washington, D. C.

Death Takes Cooper Curtice, Retired Federal Scientist

Cooper Curtice, widely known for his successful research on the life history of the cattle-fever tick and methods of eradicating it, died on August 8 at his home in Beltsville, Md., at the age of 83.



Cooper Curtice

Born in Stamford, Conn., May 7, 1856, Dr. Curtice was graduated from Cornell University with a B.S. degree in 1881, having been, as he liked to explain it, forced through school by his father, who was firmly determined that this son be an engineer. Two years later he received a D.V.S. degree from the Columbian Veterinary College in New York. Then he was graduated with an M.D. from the National Medical College, George Washington University, in 1888.

Dr. Curtice's early interest in natural history led him first into paleontology and, for three

*The U. S. Department of Agriculture offers a free leaflet, "Combating the Dog Tick," which tells how to guard dogs from tick attacks.

years, he was paleontological aid in the Geological Survey. In 1886, however, he joined the service of the federal bureau of animal industry. While an agent of the bureau he published a treatise on sheep parasites, worked out the life history of the cattle tick and promoted the study of the tick as a carrier of southern cattle fever. A free-lance worker, he seldom remained in his laboratory, preferring to study in the field.

With the exception of several periods during which he resigned to carry on other work, Dr. Curtice was in the service of the bureau until his retirement in 1930. Even after that, he remained a collaborator without compensation, working with private resources at Fairfax, Va., and McNeill, Miss., until failing health ended his professional activities two years ago.

Among other of his significant projects was his study of the control of stomach worms in sheep, which culminated in the differentiation from tuberculosis of a disease caused by this parasite, and a subsequent improved method of treating lambs for preventing this malady.

Dr. Curtice is survived by his widow and seven children.

Bureau of Foreign and Domestic Commerce Department of Commerce

UPTURN IN FOREIGN DEMAND FOR AMERICAN MEDICINES

Foreign demand for American medicinal products has inclined steadily upward during the current year, with veterinary biological products drawing a considerable share of the increment. Total exports of medicinal, pharmaceutical and biological supplies in the first half of 1939 were valued at $9,809,000 as compared with $8,250,000 in the corresponding period of 1938—a gain of almost 10 per cent.

Outstanding gains were recorded for nonproprietary veterinary biological products, shipments of which increased in value from $180,000 in the first six months of 1938 to $701,000 for the comparable 1939 period. It now seems probable that American medicinal and pharmaceutical exports during the whole of 1939 will exceed $20,000,000 in value, thus attaining the 1929 level.

B. A. I. Transfers

Norman W. Ackerman, from Buffalo, N. Y., to Milwaukee, Wis.; Wallace W. Boggie, from Fort Dodge, Iowa, to Madison, Ind.; Zeno C. Boyd, from Twin Falls, Idaho, to Raleigh, N. C.; Wilford J. Cross, from Baltimore, Md., to Richmond, Va.; Bert L. Dawson, from Kansas City, Kan., to Los Angeles, Calif.; John M. Dermody, from Chicago, Ill., to Fort Dodge, Iowa; Robustiano B. Ferro, from Buffalo, N. Y., to Kansas City, Kan.; James E. Guthrie, from Baltimore, Md., to Beltsville, Md.; Hugh H. Hervey, from San Francisco, Calif., to Los Angeles, Calif.; Irvin P. Irwin, from Oklahoma City, Okla., to Des Moines, Iowa; Dana A. La Mar, from Chicago, Ill., to El Paso, Texas; Wm. C. Logan, from San Juan, P. R., to Chicago, Ill.; E. Leland Love, from Topeka, Kan., to Beltsville, Md.; Robert A. Moody, from Charleston, W. Va., to Chicago, Ill.; Warren D. Murdock, from Little Rock, Ark., to St. Paul, Minn.; Joseph F. Nieberding, from Oklahoma City, Okla., to Atlanta, Ga.; Wm. F. Osterholtz, from Omaha, Neb., to St. Louis, Mo.; Arthur B. Rogers, from Baltimore, Md., to Raleigh, N. C.; Arthur Seligman, from Charleston, W. Va., to Baltimore, Md.; Hubert L. Smith, from Fort Worth, Texas, to Kansas City, Kan.; Calvin A. Trapkey, from Buffalo, N. Y., to Cincinnati, Ohio; Simpson M. Turner, from Omaha, Neb., to San Francisco, Calif.; Albert G. Ward, from Buffalo, N. Y., to Indianapolis, Ind.

Regular Army

Lt. Col. Harold E. Egan is relieved from assignment and duty at Fort Francis E. Warren, Wyo., effective on or about September 1, 1939, is then assigned to headquarters, seventh corps area, Omaha, Neb., and additional duty as attending veterinarian at Fort Omaha and Fort Crook, Neb.

Lt. Colonel Gerald W. Fitz Gerald is assigned to duty at San Antonio General Depot, San Antonio, Texas, and to additional duty as attending veterinarian at Randolph Field, Texas, effective upon completion of his present tour of foreign service.

Lt. Colonel Jesse D. Derrick is relieved from duty at headquarters, seventh corps area, Omaha, Neb., and from additional duty as attending veterinarian at Fort Omaha and Fort Crook, Neb., effective in time to proceed to New York, N. Y., and sail on transport scheduled to leave that port on or about September 9, 1939, for the Hawaiian department.

Announcement is made of the promotion of the following-named officers to the grade of captain with rank from date opposite their names:

1st Lt. Robert J. Brown, July 15.
1st Lt. Earl G. Kingdon, July 20.

Lt. Colonel Oscar C. Schwalm is relieved from assignment and duty at Fort Bliss, Texas, about August 24, 1939, and is assigned to duty at Fort Des Moines, Iowa.

Authority has been granted for the calling to extended active duty of a limited number of veterinary reserve officers in connection with the air corps expansion program. These officers will be assigned to duty at the larger air fields for the inspection of meat, meat-food, and dairy products.

The following veterinary officers have been designated to make inspections of meat, meat-

food, and dairy products, at the points indicated, for supply to the various units engaged in the First Army maneuvers:

Lt. Col. Irby R. Pollard, Fort Worth, Texas
Major Stanley M. Nevin, Lake Charles, La.
Lt. Col. Will C. Griffin, Chicago, Ill.
Lt. Col. Mott Ramsey, East St. Louis, Ill.

Captain Arvo T. Thompson, on duty at the Army Veterinary School, Army Medical Center, Washington, D. C., has been detailed to make inspections at the following named places in order: Beatrice, Neb.; Columbus, Neb.; Atlantic, Iowa; Clay Center, Kan.; Salina, Kan.; St. Joseph, Mo.; and Kansas City, Mo.

Veterinary Corps Reserve

PROMOTIONS

To Captain: Glen Orahood Fly, Donaldson Arms Apt., Evansville, Ind.; Vaughn Eugene Ishee, R.F.D. No. 1, Middlefield, Ohio; Geo. Miller Kerr, 8 Garfield Ave., Hyattsville, Md.; Leo Leibach Lieberman, 276 Mill Hill Ave., Bridgeport, Conn.; Andrew J. McCreary, 803 N. Palafox St., Pensacola, Fla.; Lloyd Jake Michael, 1808 Parker St., Cleveland, Tenn.

To 1st Lieut.: Ferdinand Ossian Johanson, R.R. No. 4, Newfield, N. Y.; Albert Kenneth Tice, 7 Wheeler Ave., Cortland, N. Y.

To Major: Joseph H. Kitzhofer, 332 P. O. Bldg., Oklahoma City, Okla.

NEW ACCEPTANCES—FIRST LIEUTENANTS

Abell, Leonard Jason, Rome, Pa.

Adams, Dale Sanford, R.F.D. No. 2, Sioux Rapids, Iowa.

Alson, Marion Colvert, Carthage, Mo.

Ayres, John Patrick, Box 14, Corbettsville, N. Y.

Bailey, Lucien Percy, Rock, W. Va.

Barner, Ralph David, 2740 Newbern St., Youngstown, Ohio.

Barrett, Clinton David, Spur, Texas.

Batcheldor, Geo. Dallas, Box 294, Yazoo City, Miss.

Beauchamp, John Woodrow, R.F.D. No. 8, Box 980, Bakersfield, Calif.

Bennett, Clarence Kinsey, R.F.D. No. 4, Hennessey, Okla.

Bent, Clarence Farrar, Leghorn Acres, Hudson, N. H.

Berger, Joseph Herman, R.F.D. No. 1, Box 80, Mishawaka, Ind.

Birch, Frank McKeenan, R.F.D. No. 2, Ithaca, N. Y.

Bjornson, Sidney Sigurd, 1142-12th St., N., Fargo, N. D.

Blomquist, Ernest William, Olalla, Wash.

Bly, Howard Judson, 1817 Church St., Evanston, Ill.

Boldt, Vincent Leo, 31 Ontario St., Honeoye Falls, N. Y.

Bolks, Herbert Paul, Hull, Iowa.

Bowstead, Warren Elbert, 709-11th Ave., DeWitt, Iowa.

Boyer, Marcus Earl, Lena, Ill.

Boyer, Paul Emerson, R.F.D. No. 4, Ashland, Ohio.

Branch, Lemuel Alexander, Franklin, Va.

Brooks, Willard Crawford, Box 67, Prairie Grove, Ark.

Broussard, George Patout, 417 East Main St., New Iberia, La.

Burt, Clark Eugene, 310 Campus St., Pullman, Wash.

Chapman, Lee Roy, Box 205, Roscoe, Texas.

Christian, Arthur Booth, 612 Hoffman St., Elmira, N. Y.

Christian, Paul, Box 1253, Waco, Texas.

Clinton, Robert Lester, R.F.D. No. 2, Pullman, Wash.

Clem, Wesley Allen, Jr., Randolph Road, Meadowbrook, Pa.

Clements, James Halferty, 422 E. 22d St., South, Newton, Iowa.

Coburn, George Carlton, 610 Nolan St., Big Spring, Texas.

Combs, Clarence Carl, Jr., 1214 Stewart Ave., Ithaca, N. Y.

Cooper, Harold Young, Titusville Road, Union City, Pa.

Corry, Willard Ellis, 703 W. 7th St., Little Rock, Ark.

Couch, J. B., Route No. 4, Grandview, Texas.

Crandall, Mark Robert, 116 Delaware Ave., Ithaca, N. Y.

Damer, Eugene Francis, R. F. D. No. 1, Box 208, Webb City, Mo.

Daugherty, William David, 759 E. Main St., Galesburg, Ill.

Dee, Clarence Everett, Melbourne, Iowa.

Druley, Vincent Yale, 3026 Main St., Kansas City, Mo.

Duey, Fernley Willard, Bellevue, Wash.

Dungan, William Michael, Jr., 1804 W. 39th St., Vancouver, Wash.

Eastep, Oren Gafford, 311 W. 9th St., Stillwater, Okla.

Enge, Clifford Oray, Clarion, Iowa.

Enge, Percy Clifford, Clarion, Iowa.

Evans, Milton Roscoe, 3835 E. 11th St., Long Beach, Calif.

Faatz, Gerald Almon, Weedsport, N. Y.

Fagan, Raymond, 567 Bradford St., Brooklyn, N. Y.

Fallon, Walter Joseph, Constable, N. Y.

Ferber, Robert, 33-17-145th St., Flushing, N. Y.

Finney, Elkana Malcolm, Cedarville, Ohio.

Fish, Ralph Cooper, Jr., 2nd & Center Sts., Delanco, N. J.

Fox, Charles William, R. F. D. No. 3, Sycamore, Ohio.

Gates, Morse Andrew, 228 Lincoln Ave., Hebron, Neb.

Gearhart, Benjamin Franklin, Jr., Box 456, Marfa, Texas.

Gillespie, James Howard, 937 Main St., Bethlehem, Pa.

Glass, Sherman Albert, 2769 Lancashire Road, Cleveland Heights, Ohio.

Gold, Russel Willie, 321½ Ash, Ames, Iowa.

Gorman, Lester Joseph, 309 Clermont, East Point, Ga.

Gregory, Douglas H., Box 65, Lewiston, Utah.

Hampton, Glen Grisby, Jr., Gothenburg, Neb.

Harris, Earl Merton, R. F. D. No. 3, Box 12, Klamath Falls, Ore.

Harris, William Kenneth, R. F. D. No. 2, Marengo, Ohio.

Haines, William Albertson, Jr., Edgely Hall, Bristol, Pa.

Hauge, Ronald Thompson, 3822 Miller Way, Sacramento, Calif.

Heller, Oliver Perry, Waterville, Ohio.

Henson, Benjamin Schubert, 74 E. Main St., Shelby, Ohio.

Herron, James Thomas, 124 South Central Ave., Cannonsburg, Pa.

Hoffman, William Frank, 18 N. 18th St., Harrisburg, Pa.

Holmes, John Mansfield, Lawyersville, N. Y.

Hood, James MacMillan, R. F. D. No. 1, Independence, Iowa.

Hostetler, Roy Ivan, Asotin, Wash.

Houser, Raymond Ralph, R. F. D. No. 5, Findley, Ohio.

Hovland, Reuben Bernhard, R. R. No. 1, Eagle Grove, Iowa.

Howell, Welling Clifford, Summit Ave., West Trenton, N. J.

Hoy, Lyman Lester, 127 Stone St., Carthage, N. Y.

Hutton, Aubrey Means, 231 Michigan Ave., St. Joseph, Mo.

Irwin, William Emory, R. R. No. 2, Cassville, Mo.

James, Harold David, R. F. D. No. 1, Yellow Springs, Ohio.

Jenkins, Oliver Leo, Gothenburg, Neb.

Jones, Leo Meyer, 3009 Wood St., Ames, Iowa.

Jones, Russell Claire, Jr., 322 E. Walnut St., Shillington, Pa.

Juni, Robert Philip, 4104 Taylor Ave., Drexel Hill, Pa.

Kelley, Elbert Lloyd, Utopia, Texas.

Key, Joseph Boston, R. R. No. 2, Kansas City, Kan.

Krewitz, Leonard, 4701 Ramona Ave., Philadelphia, Pa.

Labahn, Charles Davis, 127 West Culton, Warrensburg, Mo.

Lasher, Norbert Augustus, Minoa, N. Y.

Lee, Philip Arthur, 6317 Fairmont St., El Cerrito, Calif.

Leonard, George Allison, Pawnee City, Neb.

Lightbody, Hugh Maurice, 1004 Court St., Charles City, Iowa.

Lovik, Stanley Arnold, Main St., Joice, Iowa.

Luker, Charles Thomas, R. R. No. 2, Dublin, Texas.

McCamish, John Nathan, 120 E. Magnolia St., San Antonio, Texas.

McFarland, Ray William, 907 Monterey Ave., Monrovia, Calif.

McFarland, Robert James, 907 Monterey Ave., Monrovia, Calif.

McCarthy, James Joseph, Jr., R. F. D. No. 2, Plainfield, N. Y.

McKenzie, Kenneth J., Baxter, Iowa.

Mack, Ernest James, 306 N. Main, River Falls, Wis.

Martin, Jerry Tom, Mt. Pleasant, Texas.

Melworth, Samuel, 2007 W. 83rd St., Cleveland, Ohio.

Mickelson, Clayton Hileman, 947 S. W. Broadway, Portland, Ore.

Miller, Harold Kleinfelter, Cleona, Pa.

Mills, Harold Boswell, 1804 Main St., Big Spring, Texas.

Molt, Fred Lester, Hinton, Okla.

Munson, Matthew Bernard, 3009 Wood St., Ames, Iowa.

Muntz, Karl Huber, Holgate, Ohio.

Murphy, Leslie Carlton, Box 23, College Station, Pullman, Wash.

Nelson, Clifford Lloyd, Rt. 1, Essex, Iowa.

Niemeyer, Wm. Edward, 203 Adm. Bldg., Union Stock Yards, Los Angeles, Calif.

Niffenegger, Dean Lloyd, Westchester, Iowa.

Nold, Florian William, Parker, S. D.

Palen, Joseph Selverius, 105 West 16th St., Hays, Kan.

Payne, Herschel Howard, Box 3, El Campo, Texas.

Pentick, Albert Paul, Setauket, N. Y.

Poppenhouse, Curtis Albert, 416 Laramie, Manhattan, Kan.

Poppenhouse, Gerhard Charles, 416 Laramie St., Manhattan, Kan.

Povar, Ralph, 1771 Union St., Brooklyn, N. Y.

Primerano, Walter Dominic, 43 Grove Ave., New Rochelle, N. Y.

Reich, Sigmund Theodore, 106 S. Main St., New London, Conn.

Ramsey, James Durand, 1753 B. Ave., N. E., Cedar Rapids, Iowa.

Remsberg, Glen Stanley, LaHarpe, Kan.

Rieke, Glenn Warner, R. R. No. 1, Blairstown, Iowa.

Rivers, Ernest William, 3 Forest St., Stoneham, Mass.

Robertson, Harry John, Grove Ave., National Park, N. J.

Rodabaugh, Donald Edwin, Norborne, Mo.

Ross, Martin Allen, R. F. D. No. 1, Mason, Ohio.

Rovner, Bernard Barny, 1220 West Columbia Ave., Philadelphia, Pa.

Rushmore, Rowland Wayne, 309 Clermont, East Point, Ga.

Sacks, Sander Alexander, 817 So. Alden St., Philadelphia, Pa.

Sasmere, Daniel Paul, 772 East 9th St., Brooklyn, N. Y.

Schofield, Richard Clark, 60 Washington St., Wellesley Hills, Mass.

Scholle, Carl Henry, 501 West 31st Street, Bryan, Texas.

Seeley, Alpheus Heise, Fort McPherson, Ga.

Sellers, Alvin Ferner, 509 No. Center Ave., Somerset, Pa.

Sepponen, Carl Matt, R. F. D. No. 1, Box 108, Roseville, Calif.

Shoeman, Henry Paul, R. F. D. 1, Waukee, Iowa.

Shultzman, LaMont Hilton, R. F. D. No. 1, Orrville, Ohio.

Sivyer, Roland Eugene, P. O. Box 162, Vancouver, Wash.

Skelton, Daniel (Colored), 84 W. 115th St., New York, N. Y.

Smeltzer, Warren Henry, Jr., 2737 N. Kimball Ave., Chicago, Ill.

Smith, Earl Prentice, 1529 Burnett Ave., Ames, Iowa.

Smith, Sydney Murray, 1130 Taylor St., San Francisco, Calif.

Smith, Gail Bevington, R. F. D. No. 1, Venedocia, Ohio.

Spieth, Emmett Woodrow, R. F. D. No. 1, Charlestown, Ind.

Spranklin, John William, Jr., 3020 Garrison Blvd., Baltimore, Md.

Sprunger, Earl Melvin, 646 Beall St., Wooster, Ohio.

Steele, Darrell Stanley, Zionsville, Ind.

Steinbach, Karl Friedrich, 121 E. Cardinal Rd., Wildwood, N. J.

Steinmetz, William Edward, 510 Campus St., Pullman, Wash.

Stephens, Seidel Miles, Box 921, Fort Worth, Texas.

Stoudenmire, William Frank, 401 E. Minnesota Ave., De Land, Fla.

Strawn, Willard Mason, R. F. D. No. 1, Milford Center, Ohio.

Swarthout, Edward William, Route 1, Box 208, Florissant, Mo.

Tannebaum, George Gilbert, 930 Montgomery St., Brooklyn, N. Y.

Taylor, Edgar Lewis, Box 493, Tallulah, La.

Teller, Willard Bruce, 210 No. Washington, Prescott, Ariz.

Thomas, Geo. Oliver, Jr., Ringgold, La.

Tierstein, Victor, 816 So. San Pedro St., Los Angeles, Calif.

Vollmar, Roland Emil, Montgomery, Minn.

Wadsworth, Ralph Harold, 139 No. Mulberry St., Mansfield, Ohio.

Walmsley, George, 2 Garrison St., Fairhaven, Mass.

Ward, Drue Simpson, General Delivery, Valley View, Texas.

Washburn, Paul Morgan, R. F. D. No. 1, Oak Hill, Ohio.

Watts, Floyd Bentley, c/o Globe Laboratories, Fort Worth, Texas.

Weaver, Lonnie Hobson, Box 386, San Antonio, Texas.

Weston, Ralph Silas, 1 Welch St., San Rafael, Calif.

Yager, Robert Henry, Somerset, Va.

Zickefoose, Edward Bonjour, Rossville, Kan.

NEW ACCEPTANCE—MAJOR

Pyle, Norman James, 105 N. Middleton Road, Pearl River, N. Y.

Civilian Conservation Corps

Effective July 1, 1939, a new policy was adopted in connection with medical, veterinary, and dental services rendered for the CCC. As the periods for which they were called to active duty expire, reserve officers of the medical department will be replaced by physicians, veterinarians, and dentists in a civilian status. Under this plan all duty performed by medical department reserve officers on active duty for the CCC will be done by members of the three professions indicated on a civilian basis.

It is anticipated that a relatively large number of these reserve officers will accept civilian appointments.

NEW ASSIGNMENT TO ACTIVE DUTY WITH CCC

Captain Richard E. Geisler, Fort Douglas, Utah.

TERMINATION OF ASSIGNMENT TO ACTIVE DUTY WITH CCC

1st Lieutenants Ray S. Attaway, Absolom B. Rich, and George A. Morrison of Indiana District, Arizona District CCC, Phoenix, Ariz., and Fort Douglas, Utah, respectively.

In reply to a direct question from Senator Hayden of Arizona, "Is the army mule more or less passé?" Maj. Gen. Henry Gibbons, quartermaster general of the Army, said, "Well, yes sir, practically."

While Congress and the quartermaster department of the Army have decided that the army mule is no longer an important part of the military setup, it is nevertheless strange that during the invasion of Ethiopia by Italy and during the entire period of the Spanish revolution, the importation of horses was an important project. Technical reports from the forces of General Franco indicate that horses played an important part in the transportation of Franco's forces and contributed largely to his success.

AMONG THE STATES

Alabama

RABIES IN BIRMINGHAM*

Rabies is primarily a veterinary problem. The incidence in man will probably never be sufficient to classify the disease as a public health problem because it is a rare human disease, a disease not easily contracted by man. Were it as easily contracted by man as is commonly supposed, it would become one of the leading causes of death because of its prevalence in dogs.

In Birmingham, the incidence in dogs has steadily increased during the past ten years. The figures are based upon the actual number of animals examined and found to be affected with rabies. The animals affected with rabies are mostly unconfined, home-owned pets. But few homeless dogs are included. They seldom arrive for laboratory diagnosis.

For 1938, the dog population of Birmingham was 20,609. Of this number, 13,012 were owned by whites and 7,597 by negroes. Negroes constitute 40 per cent of the human population. The latter are not much concerned about rabies. Only 12.5 per cent of the dogs submitted for examination out of a total of 245 were owned by negroes. In areas where negroes own 75 to 100 per cent of the dogs the incidence of rabies (among dogs) was but 0.5 per cent. The rate becomes higher as the percentage (1.5 per cent) of white-owned increases. The discrepancy is due in part to the failure of negroes to report their rabid dogs. This disregard is shown also by studies of the effectiveness of canine vaccination. Out of 799 dogs vaccinated for negroes, not one was found rabid. On the other hand, among whites there were vaccinated 5,107 dogs and, of these, 0.5 per cent (26) were submitted for examination and found rabid.

During the ten-year period (1929-1938), 3,111 heads were examined. Of these 1,495, or 48 per cent, were positive. Among the 5,206 persons who were given the Pasteur or Semple treatment there were three deaths from rabies.

For Alabama during the 17-year period (1922-1939), 11,218 animals were positive; 42,947 persons were given antirabies vaccine; and forty-eight (0.06 per cent) died of the disease. The wide difference between the number given the treatment and the number of deaths is due to "undue apprehension" among bitten persons. Bitten persons are unwilling to accept medical advice and insist on vaccine treatment.

A significant finding in the Alabama statistics is that out of the 48 deaths, 23, or 48 per cent, were said to have received prompt and adequate treatment. Thus, the fatalities among the treated and untreated persons afford ground for speculation. On the other hand, this low rate, according to the interpretations of statistics, is a favorable showing for the treatment.

All of this work indicates that the dog is the principal animal involved—the animal directly responsible for the spread of the disease to other animals.

There was little difference in mortality between treated and untreated persons, that is to say, between the highly exposed untreated negroes and the highly exposed treated whites.

In effect, the authors believe in antirabies treatment but consider much of the treatment ill-advised. They set down in detail all of the various conditions under which treatment is or is not advisable.

* * *

ALABAMA STATE VETERINARY MEDICAL ASSOCIATION

Highlighted by a dance at the Selma Country Club and an address by the Hon. Heygood Patterson, commissioner of agriculture and industries of the State of Alabama, 75 Alabama veterinarians, many of whom were accompanied by their wives, spent July 20-21 in attendance at the midsummer meeting of the state association at Selma.

With routine business limited to a night session on the first day, J. H. Ryland presided over an excellent program comprising, to name but a few of the presentations, "Hydrogen Dioxide as a Canine Vermicide," by Leon F. Whitney, class of '40, Alabama Polytechnic Institute, Auburn; "Veterinary Ethics," by G. D. Ingram of Florence; "Equine Encephalomyelitis," by L. E. Beckman of Tuscaloosa; "The Use of Sulfanilamide," by J. T. Atkinson of Selma; and "Large Animal Practice," by A. L. Halloway of Atmore.

The two-day meeting was terminated with a barbecue dinner given by the Dallas county practitioners and a visit to the Black Belt Agricultural Experiment Station, arranged through the courtesy of Superintendent K. G. Baker.

R. L. MUNDHENK.

California

To the Editor.—The article, "Horseshoeing in Hauran," which appeared on pp. 133-136 of the August issue of the JOURNAL, amplified fond memories of my sojourn in the A.E.F. I was veterinarian in charge as well as officer in charge of a very valuable stable of Arabian horses and racing camels, the property of the

*G. A. Dennison and J. D. Dowling. Human mortality as affected by rabies treatment. Jour. A.M.A., cxiii (July 29, 1939), pp. 390-394.

Prince of Hedjaz. I have in my possession one of the oldest bits in existence and one of the very old-time horseshoes mentioned in the JOURNAL article.

The Arabian group was composed of various tribes or clans—several tribal chieftains, nobility, swordsmen, ordinary soldiers, and a few slaves.

WM. D. ODOU,
Montebello, Calif.

Comment.—Dr. (Captain) Odou (Chi. '17), formerly of North Dakota, was a prominent figure of the veterinary corps of the World War. He served in the veterinary liaison service in Paris during the major operations and post-Armistice period, when the disposal of American mounts became an intricate, international project.

Canada

CENTRAL CANADA VETERINARY ASSOCIATION

A meeting of the Central Canada Veterinary Association was held at Brockville, Ont., August 4-5, 1939. Over 100 veterinarians were registered from Ontario, Quebec, the states of Michigan, Vermont and New York.

B. T. McGhie, deputy minister of health for the province of Ontario, addressed the gathering. A well attended clinic was held the last day of the meeting at the Avondale farm, owned by Senator Hardy. H. J. Milks of Cornell University, Ithaca, N. Y., officiated as surgeon at this event.

W. MOYNIHAN, *Resident Secretary.*

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ONTARIO VETERINARY ASSOCIATION

The midsummer meeting of the Ontario Veterinary Association was held on July 11-13, 1939, at Guelph, Ont. All present agreed that this was one of the most successful veterinary conferences ever held in Ontario.

Papers on various subjects of current importance were presented and discussed. A banquet was held on the evening of the 13th in the dining room of the Royal Hotel at Guelph. President H. R. Potter of Niagara Falls introduced the guest speaker, Mr. Blake Duff, who gave a splendid address on the significance of the visit of the King and Queen to Canada. Visitors from the states of Michigan and New York were present.

W. MOYNIHAN, *Resident Secretary.*

Colorado

A. A. Hermann (Colo. '10) of Denver won two cash prizes for himself and Henry Hoffman, manager of the Hermosa Hospital in Denver, at the New York World's Fair on August 20 for writing one of the four best letters out of thousands in the weekly "Letters Home" contest at the exhibit of the Westinghouse Electric and Manufacturing Company. Ray Perkins, radio star, awarded the prizes on a coast-to-coast NBC broadcast in which Dr. Hermann appeared and read an excerpt from his letter. Dr. Hermann was visiting the East before beginning the trip to the A. V. M. A. convention.

A. A. Hermann (right) receives his award in the "Letters Home" contest. Ray Perkins, radio star, is making the presentation.

District of Columbia

DISTRICT OF COLUMBIA
VETERINARY MEDICAL ASSOCIATION

The District of Columbia association held its third quarterly meeting of the year at the Mayflower Hotel, Washington, D. C., on the evening of July 18, H. W. Schoening presiding.

Lt. Col. Raymond Randall gave a very interesting paper on "Preparation of Equine Encephalomyelitis Vaccine (Chick) at the Army Veterinary School for Use on Army Horses." Lt. Col. R. A. Kelser, director of the U. S. veterinary corps, opened the discussion of this paper. It was pointed out in the discussion that by taking every precaution before vaccinating the horses, no severe reactions were noted in the 39,000 animals given the encephalomyelitis vaccine prophylaxis. Motion pictures on the preparation of equine encephalomyelitis vaccine (chick) concluded the program.

WM. M. MOHLER, *Secretary.*

Florida

The only farm that breeds pedigreed quail is located at Green Grove Spring, Fla. The farm has 5,000 breeding quail, 785 breeding chukars, and 10,000 pheasants. Its output is shipped to all parts of the United States and Central and South America.

Idaho

IDAHO VETERINARY MEDICAL ASSOCIATION

The summer meeting of the Idaho association was held at Idaho Falls, June 26-27, 1939, K. K. Shott of Buhl presiding.

Many excellent scientific papers were presented and, on the afternoon of the first day and the morning of the second day, a clinic was held. The outstanding feature of this portion of the program was an illustrated discussion of fractures in small animals by J. C. Flint of Salt Lake City, Utah.

On the evening of the 26th, a banquet was held. Dr. E. H. Ikard of Gooding acted as toastmaster. Forty-three veterinarians and their wives were in attendance.

This was considered to be one of the most thoroughly planned meetings in the history of the Association, although the attendance was not as large as it might have been had the meeting been held in a city more centrally located. The local committee on arrangements and the members of the Association who participated are to be commended for their fine spirit of coöperation.

E. M. GILDOW, *Secretary-Treasurer.*

Indiana

The veterinarians of Indiana will honor R. A. Craig (Iowa '97), director of the veterinary department of Purdue University, during the annual meeting of the Indiana Veterinary Medical Association at Indianapolis, January 9-11, 1940. A portrait of Dr. Craig painted by a nationally known Indiana artist, will be unveiled and presented to President Elliott of Purdue University at the meeting.

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The *Poultry Tribune* (August, 1939) devotes a full column to the report of the Second Nutrition Conference for Veterinarians, held at Decatur and Fort Wayne on July 12 under the sponsorship of the Central Soya Company, Inc.

Iowa

SWINE SECTION, EASTERN IOWA CLINIC

The swine section of the Eastern Iowa Veterinary Association's clinic, held at Waterloo, July 20, was directed by Chairman McGrath of Elma, T. J. Gilloon of Dyersville, W. L. Hanson of Parkersburg, and J. W. Giffee of the hog-cholera-control division, U. S. bureau of animal industry, Cedar Rapids. The latter presided at the autopsy table and employed an approved technic for making complete postmortem examinations. The other members of the committee assisted and kept the clinic sheets. Practitioners who presented the subjects supplied the histories, and various members of the laboratory section checked the lesions and contributed to the discussions.

The large number of subjects presented were all in the young pig class, the animals ranging from 3 to 9 weeks of age. If it be logical to appraise the ailments of young pigs most troublesome to practitioners in eastern Iowa, there can be but one conclusion: Soil-borne diseases predominate. Enteritis, parasitisms and pulmonary pathology easily occupied the major portion of the program and constituted a convincing argument in favor of the attention research workers accord soil-borne diseases of swine.

It is noteworthy that no surgical cases were presented at this clinic, since at previous clinics of the Association many surgical exercises were demonstrated.

JOHN B. BRYANT, *Associate Editor.*

Massachusetts

Through the efforts of the Massachusetts Veterinary Association a change in the veterinary practice act of the state was effected on June 1. The amendment to the act provides for the existence of an approving authority consisting of the commissioner of education, the director of the division of livestock disease control of the state department of agriculture, and the secretary of the state board of registration in veterinary medicine.

It is provided that this approving authority shall pass on the qualifications of applicants to take the state board examinations. The act further provides that applicants shall be graduates of colleges with five-year courses which meet the requirements of this approving authority.

Missouri

MISSOURI VETERINARY MEDICAL ASSOCIATION

An excellent program of timely subjects marked the summer meeting of the Missouri association, held at St. Louis, June 15-16, 1939.

Infectious abortion in cattle and its relation to human diseases was the subject of a paper presented by O. E. Hagenbusch, M. D., of St. Louis. It prompted a lively discussion, as did the paper on human encephalitis and its relation to encephalomyelitis by T. P. Pote of the Washington University medical school, St. Louis.

M. R. Fisher, chief of milk control in the City of St. Louis, discussed the veterinarian and his local milk-control regulations, citing the significance of veterinary assistance in this phase of public health work.

The fact that both the veterinarians and the physicians in attendance engaged spiritedly in the discussions indicates the development of closer coöperation between physicians and veterinarians in public health programs.

J. L. WELLS, *Secretary-Treasurer.*

Montana

MONTANA VETERINARY MEDICAL ASSOCIATION

Forty veterinarians from all parts of the state gathered at Bozeman on June 30-July 1 for the 31st annual meeting of the Montana

association. Clyde McKee, director of the Montana Agricultural Experiment Station at Bozeman, delivered the address of welcome, and J. W. Kilpatrick of Hamilton responded. W. J. Butler of Helena presided.

The following men contributed to the program: B. K. Kilbourne, epidemiologist of the state board of health, Helena; C. L. Heath of Billings; J. D. C. Wipf of Bozeman; H. Marsh of Bozeman; A. L. Strand, president of the Montana State College, Bozeman; C. B. Philip of the Rocky Mountain Laboratory, Hamilton; Fred F. McKenzie, in charge of physiology of reproduction in Equidae at the Range Livestock Experiment Station, U. S. Department of Agriculture, Miles City, assisted by John Cummings of the animal husbandry department of Montana State College; W. J. Butler, state veterinarian, Helena; and G. W. Cronen, inspector in charge, U. S. bureau of animal industry.

A business meeting concluded the convention. Helena was selected as the site for the 1940 meeting. B. O. Fisher of Great Falls was elected to the presidency for the ensuing year. W. E. Logan of Billings was chosen vice-president, and E. A. Tunnicliff of Bozeman was reëlected secretary-treasurer.

E. A. TUNNICLIFF, *Secretary-Treasurer.*

New Jersey

The New Jersey Veterinary Medical Association has been invited to participate in the program of the New Jersey Health Officers Association in November of 1939. Earlier in the year, the state association, at the request of the agricultural groups, took an active part in the program presented during Agricultural Week.

New York

Overheard in the lobby of the hotel headquarters of the recent meeting of the New York State Veterinary Medical Society was a discussion of an incident of unwise use of live culture abortion vaccine. A herd of purebred cows about to be sold at a public sale were given an injection of abortion vaccine (live culture) without any rational reason for doing so. When tested after the sale, most of the cows reacted and could not be delivered to out-of-state buyers, notwithstanding that the herd was known to be abortion-free before being vaccinated.

Tennessee

According to A. C. Topmiller, state veterinarian, there were 71 outbreaks of hog cholera in 20 counties of Tennessee during the month of July, with eleven outbreaks being reported in one county—Sumner. There were 18 outbreaks of erysipelas in 14 counties and four of hemorrhagic septicemia in three counties.

COMING MEETINGS

Small Animal Hospital Association. Los Angeles, Calif. September 5, 1939. R. W. Gerry, Secretary, 8474 Melrose Ave., Los Angeles, Calif.

Dallas-Fort Worth Veterinary Medical Society. Adolphus Hotel, Fort Worth, Texas. September 7, 1939. H. V. Cardona, Secretary, 2736 Purington Ave., Fort Worth, Texas.

Chicago Veterinary Medical Association. Chicago, Ill. September 12, 1939. W. A. Young, Secretary, 157 W. Grand Ave., Chicago, Ill.

Saint Louis District Veterinary Medical Association. Melbourne Hotel, Saint Louis, Mo. September 13, 1939. J. P. Torrey, Secretary, 610 Veronica Ave., East Saint Louis, Ill.

Willamette Valley Veterinary Medical Association. Albany, Ore. September 13, 1939. T. Robert Phelps, Secretary, Oregon City, Ore.

Kansas City Veterinary Medical Association. Kansas City, Mo. September 18, 1939. S. J. Schilling, Secretary, Box 167, Kansas City, Mo.

San Diego County Veterinary Medical Association. Zoölogical Research Bldg., Balboa Park, San Diego, Calif. September 18, 1939. Glenn A. Tucker, Secretary, Vista, Calif.

Southern California Veterinary Medical Association. Chamber of Commerce Bldg., Los Angeles, Calif. September 20, 1939. Charles Eastman, Secretary, 725 S. Vancouver Ave., Los Angeles, Calif.

Massachusetts Veterinary Association. Boston, Mass. September 27, 1939. H. W. Jakeman, Secretary, 44 Bromfield St., Boston, Mass.

Southern Alabama—Western Florida Veterinary Medical Club. Mobile, Ala. September 29, 1939. E. M. Nighbert, Secretary, Cantonment, Fla.

New England Veterinary Medical Association. Hotel Bond, Hartford, Conn. October 2-3, 1939. H. W. Jakeman, Secretary, 44 Bromfield St., Boston, Mass.

Veterinary Medical Association of New York City. Hotel New Yorker, New York, N. Y. October 4, 1939.

Houston Veterinary Association. Houston, Texas. October 5, 1939. W. T. Hufnall, Secretary, 1612-14 E. Alabama Ave., Houston, Texas.

University of Illinois Veterinary Conference. University of Illinois, Urbana, Ill. October 5-6, 1939. Robert Graham, Secretary, University of Illinois, Urbana, Ill.

West Virginia Veterinary Medical Association. Kanawha Hotel, Charleston, W. Va. October 9-10, 1939. J. H. Rietz, Secretary-Treasurer, Oglebay Hall, West Virginia University, Morgantown, W. Va.

Southeastern Michigan Veterinary Medical Association. Medical Arts Bldg., 3919 John R St., Detroit, Mich. October 11, 1939. F. D. Egan, 17422 Woodward Ave., Detroit, Mich.

Maine Veterinary Medical Association. Orono, Me. October 12, 1939. A. E. Coombs, Secretary, 1 Kennebec St., Skowhegan, Me.

American Public Health Association. William Penn Hotel, Pittsburgh, Pa. October 17-20, 1939. Reginald M. Atwater, Executive Secretary.

District of Columbia Veterinary Medical Association. Mayflower Hotel, Washington, D. C. October 17, 1939. W. M. Mohler, Secretary, 5508 Nebraska Ave. N. W., Washington, D. C.

Pennsylvania State Veterinary Medical Association. Harrisburg, Pa. October 19-20, 1939. James F. Shigley, Corr. Secretary, State College, Pa.

Mississippi Valley Veterinary Medical Association. Hotel Custer, Galesburg, Ill. November 15-16, 1939. L. A. Gray, Secretary, Bushnell, Ill.

PERSONAL NOTES

Birth

To Dr. (Colo. '35) and Mrs. Wm. A. Goodfellow of Sheridan, Wyo., a daughter, Marion Delores, August 7, 1939.

Marriage

George E. Bowler (Mich. '36) of Ann Arbor, Mich., to Gladys Roberts of Jamaica, Long Island, at New York City, June 17, 1939.

Activities

David A. Walker (Corn. '32) of Morrisville, Vt., has been appointed extension animal pathologist of his state.

Willard M. Strawn (O.S.U. '39), formerly of Marysville, Ohio, has taken over the practice of the late Carl G. Vollmer and is located at 5557 Secor Road, Toledo, Ohio.

J. L. Boydston (Mich. '37), formerly of Athens, Mich., is now associated with A. J. Kline (Ont. '94) at Wauseon, Ohio.

E. G. Boydston (Mich. '35) is now located at Athens, Mich.

J. R. Peters (O.S.U. '31), formerly of Wauseon, Ohio, is now practicing in Archbold, Ohio.

George E. Bowler (Mich. '30) is now located at 322 N. State St., Ann Arbor, Mich.

Leonard J. Goss (O.S.U. '34) has replaced Chas. R. Schroeder (Wash. '29) as veterinarian for the New York Zoölogical Park, New York City. The latter is now veterinarian for the San Diego (Calif.) Zoo.

L. G. Soderholm (Wash. '35) is now engaged in general practice at Woodburn, Ore.

J. L. Wright (U.P. '32), formerly of La Grange, Ga., is now practicing in Memphis, Tenn. His hospital, the Memphis Small Animal Clinic, is located at 733 E. Parkway South.

Ray Hoefling (Cin. '11) of Cincinnati, Ohio, suffered a severe leg injury recently when he slipped while conducting a postmortem examination. Dr. Hoefling, who is attached to the meat inspection corps of the B.A.I., is reported by Otto Hornlein (McK. '10) of Chicago Ill., to be making a satisfactory recovery in the Deaconess Hospital in Cincinnati.

DEATHS

John F. McKenna of Los Angeles, Calif., died on August 16, 1939, of a pulmonary carcinoma. A graduate of the San Francisco Veterinary College, class of 1911, Dr. McKenna practiced for some time at Fresno. In recent years, however, he conducted the Ambassador Animal Hospital in Los Angeles. He joined the national association in 1912.

Teodulo T. Topacio of Manila, Philippine Islands, died on June 19, 1939, of apoplexy brought about by high blood pressure. At the time of his death he was chief of the veterinary research division of the Philippine Bureau of Animal Industry, in which position he pioneered many far-reaching disease-control measures.

Dr. Topacio studied from 1905 to 1909 in the University of Nebraska, where he received a B.S. degree. In 1912, he entered the College of Veterinary Science, University of the Philippines, from which he was graduated in 1915. In 1927, Dr. Topacio returned to the United States to continue his studies in the State College of Washington. He received the degree of master of science in bacteriology the following year. Then, he took graduate work in bacteriology and pathology at the University of Pennsylvania. He studied from 1930-31 at the Johns Hopkins University school of hygiene and public health, receiving the degree of doctor of science in hygiene. His doctorate thesis dealt with filtrable viruses.

Dr. Topacio joined the A.V.M.A. in 1919.

Arthur S. Makin of Birmingham, Ala., died at his home at the age of 46 on May 14, 1939, of a heart attack.

Born in Birmingham, December 8, 1892, Dr. Makin was graduated from the Alabama Polytechnic Institute in 1914. While in college, he played on the varsity football team and was a member of Delta Pi Sigma. Prior to his death, he was engaged in the service of the U. S. bureau of animal industry.

Dr. Makin joined the A.V.M.A. in 1918.

Fourteen Years

have elapsed since Lockhart and associates announced the development of Homologous Anti-Canine Distemper Serum and Antigen (Virus). (See August, 1925, Journal of American Veterinary Medical Association, and August, 1925, Veterinary Medicine.)

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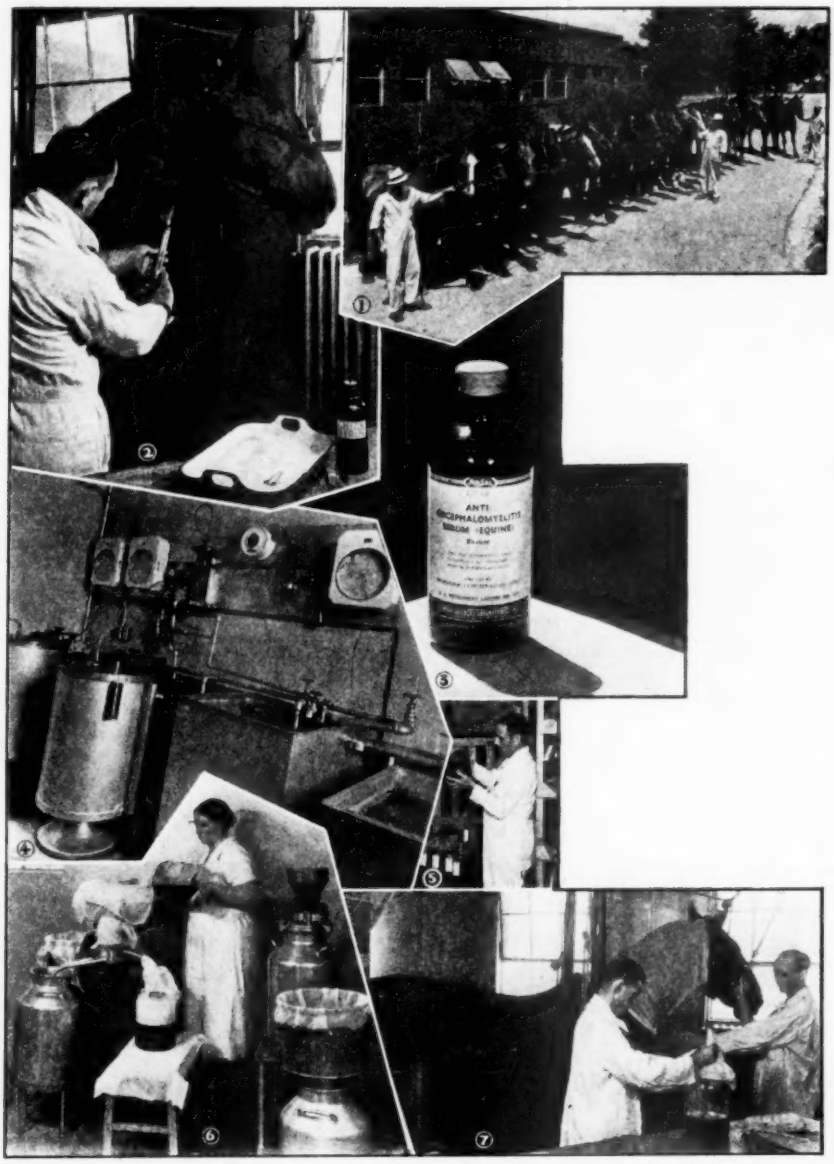

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